THE AMERICAN SURGEON

VOLUME 26

Baltimore, Maryland
1960

CONTENTS

No. 1, January 1960

The Differential Diagnosis of Intestinal Obstruction in the Newborn. David B. Sheldon, M.D.. 1
The Use of Hypothermia in Septic Shock. John N. Allen, M.D., James T. Estes, M.D., and Arlie R. Mansberger, Jr., M.D. 11
The Mechanism of Uterine Bleeding in the Presence of Fibromyomas. Narinder Sehgal, M.B.B.S., and Arthur L. Haskins, M.D. 21
Spontaneous Rupture of the Esophagus. Ralph D. Mahon, Jr., M.D., and Leonard J. Robison, M.D. 24
The Use of Hypnoanalgesia in the Relief of Intractable Pain. Maurice M. Tinterow, M.D., F.A.C.A., F.I.C.S. 30
The Surgical Significance of Rib Exostoses. Edward R. Hipp, Jr., M.D. 35
Mesenteric Cysts. Norman F. Sprague, Jr., M.D. 42
Anterior Excision of Ruptured Cervical Intervertebral Disc and Interbody Fusion (Cloward). Ralph M. Stuck, M.D., Robert F. Hall, M.D., and E. Howard Fralick, M.D. 50
Pleural and Pericardial Biopsy. William A. Hopkins, M.D., Alfredo Alarcon, M.D., M. Bedford Davis, M.D., and William C. Wansker, M.D. 57
Editorial. Warfield M. Firor, M.D. 63
Book Reviews 64

No. 2, February 1960

Postoperative Care after Cardiopulmonary Bypass for Open Heart Surgery. Robert S. Cartwright, M.D., and George J. Magovern, M.D. 65
Some Unusual Tumors of the Stomach. Charles C. Rutledge, M.D. 73
Cataract Surgery. James P. Rigg, M.D., and Robert W. Rigg, M.D. 76
A Rational Approach to the Thyroid Malignancy Problem. Henry Goodwin Glass, M.D., George W. Waldron, M.D., Herbert C. Allen, Jr., M.D., and Wilson G. Brown, M.D. 81
The Endocrinological Treatment of Advanced Breast Cancer. Robert A. Huseby, M.D. 87
Primary Tracheal Tumors. Charles Maxfield Parrish, M.D., and Robert E. Jones, M.D. 95
Post-traumatic Intussusception in Adults. John A. Dixon, M.D. 99
Lesions of the Terminal Ileum and Colon Simulating Acute Appendicitis. Robert Woodruff, M.D., and Albert E. James, M.D. 102
Mesenteric Vascular Occlusion. John D. Olson, M.D., and Frank M. Lockwood, M.D. 106
The Problem of the Common Duct Stone: Transduodenal Sphincterostomy versus Choledochotomy. Ralph R. Coffey, M.D. 110
Wound Healing. III. Artificial Maturation of Arrested Regenerate with an Acetylated Amino Sugar. B. L. Reynolds, M.D., Theodore F. Leveque, M.D., and Robert W. Buxton, M.D. 113
Surgical Treatment of Carcinoma of the Thyroid. Joseph P. McNeill, M.D., and John V. Goode, M.D. 118
Book Reviews 121

No. 3, March 1960

Submucosal Hemorrhoidectomy. W. Silen and W. B. Brown 123
Familial Polyposis Limited to Females of One Family for Three Generations. John B. Farley, M.D., Ralph M. Wexler, M.D., L. J. Farabaugh, M.D., and F. E. Stander, M.D. 129
Respiratory Carrier Turnover of Potential Pathogens in a University Hospital Surgery Department. Jerome J. Landy, M.D., Jerome Rosenfeld, M.S., Isabelle Havens, M.S., and Ross S. Benham, Ph.D. 132
Spontaneous Rupture of Hypernephroma Simulating the Acute Surgical Abdomen. Joe T. Gilbert, M.D. 136
Trackdown: The Economics of Multiple Operations for Recurrent Cancer. E. W. Czerny, M.D., and H. D. Cogswell, M.D. 141
Lacerations and Incisions: Technical Considerations. Douglas W. Macomber, M.D. 145

The Effect of Treatment for Chronic Anemia on Respiratory Dynamics. K. O. ADWAN, M.D., AND B. J. WILSON M.D. ... 152
Plastic Repair of Exstrophy of Bladder Combined with Bilateral Osteotomy of the Ilia: Progress Report. W. G. SHULTZ, M.D. ... 158
Acute Phlegmonous Gastritis. L. H. WILKINSON, M.D., A. G. SIMMS, II, M.D., AND V. T. FLOYD, M.D. ... 160
Acute Hyperparathyroidism: Case Report. R. C. DERBYSHIRE, M.D., AND RICHARD M. ANGLE, M.D. ... 166
Diverticular Disease of the Colon with Hemorrhage: A Study of 78 Cases. WILLIAM C. QUINN, M.D. 171
Some Results after Hepatocholangiojejunostomy with Roux Y. JAMES A. KIRTLEY, JR., M.D. ... 175
Book Reviews ... 180

No. 4, April 1960

Foreword. ALFRED BLALOCK, M.D. ... 183
The Heroic Treatment of Acute Head Injuries: A Critical Analysis of the Results. A. EARL WALKER, M.D., AND PERRY BLACK ... 184
Retrograde Perfusion of the Coronary Sinus with Gaseous Oxygen. JAMES L. TALBERT, M.D., LEE H. RILEY, JR., M.D., DAVID C. SABISTON, JR., M.D., AND ALFRED BLALOCK, M.D. ... 189
Hypertrophic Pyloric Stenosis in Adults. EDWARD S. STAFFORD, M.D. ... 193
Two Unusual Urethroplasties. WILLIAM WALLACE SCOTT, M.D. ... 196
Anatomical Variations in 46 Patients with Congenital Aortic Stenosis. FRANK C. SPENCER, M.D., CATHERINE A. NEILL, M.D., LEWIS SANK, AND HENRY T. BAHNSON, M.D. ... 204
Coronary Endarterectomy. DAVID C. SABISTON, JR., M.D. ... 217
Experiences with Open Heart Surgery During Cardiopulmonary Bypass in 270 Cases. HENRY T. BAHNSON, M.D., FRANK C. SPENCER, M.D., ROBERT A. GAERTNER, AND DONALD W. BENSON, PH.D. ... 227
Six Case Histories Demonstrating the Feasibility of Partial or Total Replacement of Vertebral Bodies by Bone Grafts. D. BOLING, M.D., D. TAXDAL, M.D., AND R. A. ROBINSON, M.D. ... 236
Iatrogenic Intraperitoneal Dissemination of a Fibrosarcoma of the Abdominal Wall. JAMES R. CANTRELL, M.D., AND LEE H. RILEY, JR., M.D. ... 248
A Method of Marking for the Surgical Specimen. ROBERT G. CHAMBERS, M.D. ... 257
The Correction of Deformity in the Adult Cleft Palate Patient. MILTON T. EDGERTON, M.D., F.A.C.S. ... 258
Duodenal Diverticulum: Clinical Significance and Surgical Treatment. J. C. HANDELSMAN, M.D., G. MURPHY, M.D., AND R. FISHBEIN, M.D. ... 272
The Hormone Treatment of Advanced Breast Cancer: Comparative Therapy with Testosterone and Two Experimental Androgens. EDWARD F. LEWISON, M.D., JOHN L. GROW, M.D., AND FRANCES H. TRIMBLE, M.D. ... 278
The Differential Diagnosis of Early Jaundice. C. R. LOMBARDO, M.D., AND G. P. MURPHY, M.D. ... 284
The Present Status of Surgical Treatment of Pathologic Affections of the Thyroid Gland. WILLIAM F. RIENHOFF, JR., M.D. ... 288
Hemipelvectomy After 68 Years. MARK M. RAVITCH, M.D., AND RANSOM R. BUCHHOLZ, M.D. ... 309

No. 5, May 1960

Fractures of the Hip: A Method for More Positive Stabilization—Preliminary Report of 15 Cases. BEN F. PERRY, M.D. ... 313
The Use of Urea in Intracranial Surgery. HERBERT S. BELL, M.D., AND JAMES G. ARNOLD, JR., M.D. ... 322
Cholelithic Intestinal Obstruction: Report of Four Cases. JOHN CARPATHIOS, M.D. ... 326
Xiphoid-Interposition: A Technical Modification for the Repair of Pectus Excavatum. FRANCIS ROBICSEK, M.D., PAUL W. SANGER, M.D., FREDERICK H. TAYLOR, M.D., AND ROBERT E. STAM, M.D. ... 329
Inadvertent Primary Resection for the Complications of Sigmoid Diverticulitis. H. CLINTON DAVIS, M.D., WILLIAM A. SHAVER, M.D., AND IRWIN S. MORSE, M.D. ... 332
Internal Mammary Lymph Node Biopsy as a Guide to Postmastectomy Radiation Therapy in Breast Carcinoma. EVERARD F. COX, M.D., AND ROBERT W. BUXTON, M.D. ... 335
Abdominal Symptoms from Obscure Urologic Conditions in Childhood. JOHN D. YOUNG, JR., M.D., AND MARCELLO C. LIMA ... 341

An Appraisal of the Routine Postoperative Use of Pantothenyl Alcohol for Prevention of Intestinal Atony. TIMOTHY A. LAMPHIER, M.D. 350
Experiences with the Robertson Urine Test. ROBERT G. CHAMBERS, M.D. 355
Surgical Treatment of Twenty Patients with Gross Hemorrhage from Presumed Diverticular Disease of the Colon. WILLIAM C. QUINN, M.D. 357
Perforated Esophagus: An Acute Emergency. VICTOR F. DOIG, M.D. 361
A Comparison of Shock from Massive Soft Tissue Wounds and Shock Induced by the Intravenous Injection of Gram-Negative Endotoxin. ARLIE R. MANSBERGER, JR., M.D., AND HENRY WISE, CAPT., MC 367
Editorial. SAMUEL F. MARSHALL, M.D. 377
Book Reviews 378

No. 6, JUNE 1960

Foreword. HARRY L. ARNOLD, JR., M.D. 381
Surgery in Hawaii, 1916–1960. J. E. STRODE, M.D. 382
Total Gastrectomy. J. E. STRODE, M.D. 384
A Technique of Profundus Tendon Repair: Preliminary Report. C. M. BURGESS, M.D. 391
Gastric Carcinoma: Multiple Visceral Resection in an Unusual Case of Long Term Survival. JAMES W. CHERRY, M.D. 393
Conversion of Gastrojejunostomy to Gastroduodenostomy in Treating the Dumping Syndrome. JAMES W. CHERRY, M.D. 396
Lipothymoma with Cystic Lymphangioma: Case Report. NIALL M. SCULLY, M.D. 400
Diverticulitis of the Cecum: Preoperative Diagnosis. R. G. RIGLER, M.D., AND J. W. CHERRY, M.D. 405
The Preoperative Medical Examination. ALFRED S. HARTWELL, M.D. 409
Glaucoma as a General Surgical Complication. T. W. COWAN, M.D. 411
Acute Glaucoma: Delayed Diagnosis. T. P. FRISSELL, M.D. 414
Some Pitfalls of Surgical Gynecology. H. E. BOWLES, M.D. 416
Experiences with Traumatic Subdural Hematoma. JOHN J. LOWREY, M.D. 419
Emotional Preparation for Hospitalization. HAROLD M. SEXTON, M.D. 422
Appendicitis during Pregnancy. R. T. WEST, M.D. 425
Prostatic Obstruction in Hawaii: Surgery and Ethnic Distribution. WALTER S. STRODE, M.D. 428
Twenty-Five Hundred Proctoscopic Examinations. G. C. FREEMAN, M.D. 431
Fascia Lata Grafts in Inguinal Hernia Repair. J. E. STRODE, M.D. 435
Book Reviews 441

No. 7, JULY 1960

Predatory Fish Bites in South Florida. JOHN HARRINGTON TANOUS, M.D., F.I.C.S., AND PAUL ILANO, M.D. 443
Apparatus for Hypothermic Perfusion. NAZIH ZUHDI, M.D., BILLY MCCOLLOUGH, M.D., NANCY CRAIG, M.D., GARMAN KIMMELL, M.S., JOHN CAREY, M.D., AND ALLEN GREER, M.D. 446
Ligation and Excision of the Indirect Inguinal Hernial Sac: An Inadequate Procedure in the Adult. H. U. STEPHENSON, M.D., AND T. S. RAIFORD, M.D. 451
Lacerating Wound of Heart: Report of a Case. JOSEPH HODGE, M.D., G. B. HODGE, M.D., AND W. C. HAGGERTY, M.D. 454
Multiple Amebic Granuloma of the Large Intestine: Report of a Case. W. EDWARD FRENCH, M.D., F.A.C.S. 457
Acute Right Lower Abdominal Pain in Patients with Past History of Appendectomy. JULES CAHAN, M.D., AND ALEC HORWITZ, M.D., F.A.C.S. 460
Myasthenia Gravis Associated with Benign Thymoma. EARL BELLE SMITH, M.D., F.A.C.S. 466
Experiences with the Surgical Treatment of Inguinal Hernia in the Child. CHARLES E. DAVIS, JR., M.D. 470
A Comprehensive Study of Incarcerated and Strangulated Hernias. ROY S. BUCY, M.D. 476
Jejunal Obstructions. R. A. MARTORELL, M.D., F.A.C.S. 481
Analysis of Ventilatory and Respiratory Function after 82 Thoracic and Nonthoracic Operations. ROBERT G. ELLISON, M.D., DAVID P. HALL, M.D., ROBERT E. TALLEY, B.S., AND LOIS T. ELLISON, M.D. 485
Effects of Hydroxyzine Hydrochloride on Ventricular Fibrillation in Deep Hypothermia. SIDNEY BIRDSONG, M.D., AND JAMES W. PATE, M.D. 492

Intestinal Pneumatosis. J. W. Welch, M.D., C. A. Hellwig, M.D., and Charles Pokorny, M.D... 494
Spontaneous Postoperative Expulsion of Spleen Through Appendectomy Incision. Herman Charache, M.D... 499
Clinical Evaluation of the Chvostek Sign. Elmer Hoffman, M.D... 501
Book Reviews... 506

No. 8, August 1960

An Analysis of 1500 Breast Biopsies. Francis M. Massie, M.D., and James T. McClellan, M.D.. 509
A Statistical Analysis of Cholecystectomies in a Charity Hospital. Robert O'Neal Chadwick, M.D... 516
Experiences with Acrylic Plastic for Cranioplasties. Paul J. Ross, M.D., and Franklin Jelsma, M.D... 519
Arterial Lesions of the Upper Extremity: A Clinical Review With Illustrative Cases, James D. Hardy, M.D... 525
Surgical Treatment of Hyperparathyroidism. Frank Glenn, M.D... 548
Malignant Granular-Cell Myoblastoma: Report of A Case and Review of Literature. D. T. Hunter, Jr., M.D., and J. P. Dewar, M.D... 554
The Surgical Treatment of Spasmodic Torticollis. Alexander T. Bunts, M.D... 560
Restoration of Finger Flexion with Homologous Composite Tissue Tendon Grafts. Erle E. Peacock, Jr., M.D... 564
Surgical Axioms. Edward F. Skinner, M.D... 572
Pancreatitis. Leonard A. Bible, M.D... 573
Book Review... 576

No. 9, September 1960

Carcinoma of the Breast at the University of Oklahoma, 1948–1952. James A. Webb, M.D... 577
Management of Exophthalmos. William H. Conklin, M.D., John C. McClintock, M.D., Donald H. Baxter, M.D., and Fremont C. Peck, Jr., M.D... 582
Resection of the Carotid Arteries for Neoplastic Invasion with Maintenance of Circulation. Alfred M. Keirle, M.D., and William A. Altemeier, M.D... 588
Acute Postoperative Pancreatitis. J. Harvey Johnston, Jr., M.D... 595
Recurrent Mitral Stenosis after Mitral Valvotomy: Surgical Considerations. Denton A. Cooley, M.D., and Paul R. Ellis, Jr., M.D... 601
Surgical Management of the Patient with Undiagnosed Massive Upper Gastrointestinal Tract Bleeding. T. B. Patton, M.D... 605
Ten-Year Experience with Operative Cholangiography. Meade C. Edmunds, Jr., M.D., John M. Emmett, M.D., and W. Douglas Clark, M.D... 613
The Surgical Approach to Intrathoracic Goiter. George H. Bunch, Jr., M.D... 622
The Mimicry of Tumors of the Spinal Cord. Alexander T. Bunts, M.D... 630
Editorial. John H. Mulholland, M.D... 634
Book Review... 636
Announcement... 637

No. 10, October 1960

Spinal Cordectomy in the Management of Spastic Paraplegia. Edmund A. Smolik, M.D., Francis P. Nash, M.D., and Otakar Machek, M.D... 639
Problems in the Management of Hyperthyroidism. John C. McClintock, M.D., William H. Conklin, M.D., and Donald H. Baxter, M.D... 646
One-Stage Ileostomy and Total Coloproctectomy for Ulcerative Colitis. Jack Greenfield, M.D... 649
Traumatic Diaphragmatic Hernia. Charles D. Knight, M.D., and Walter W. McCook, M.D.... 656
Smooth Muscle Tumors of the Gastrointestinal Tract. Robert W. Buxton, M.D... 666
Intra-Arterial Radioisotopes to Treat Cancer. Edgar D. Grady, M.D., Walter Sale, M.D., William Perrin Nicolson, Jr., M.D., and Luther C. Rollins, M.D... 678
Subtotal Gastrectomy for Benign Gastroduodenal Ulceration in the Patient over Sixty. William T. Williams, M.D... 685
Editorial. George Crile, Jr., M.D... 692

No. 11, November 1960

The Value of Ammonia Determinations on Peritoneal Fluid in the Differential Diagnosis of Pathologic States Resulting in Hemorrhagic Ascites: Preliminary Report. Arlie R. Mansberger, Jr., M.D., and Phillip Lankford, B.S. ... 695
Tetanus in Louisville. William H. Bizot, M.D. ... 702
Massive Gastrointestinal Hemorrhage due to Hereditary Hemorrhagic Telangiectasia. J. Richard Amerson, M.D., and William C. McGarity, M.D. ... 707
Clinical Applications of Blood Volume Determinations in Surgery. A. H. Letton, M.D., and John P. Wilson, M.D. ... 713
Value of Operative Cholangiography. Louis T. Palumbo, M.D., and Howard R. Porter, M.D. ... 723
The Use of Hypnotic Anesthesia for Major Surgical Procedure. Maurice M. Tinterow, M.D. ... 732
Iatrogenic Arteriovenous Fistula: Report of a Case Involving the Superior Mesenteric Vessels. Edward R. Munnell, M.D., Carlos R. Mota, M.D., and William Best Thompson, M.D. ... 738
Surgical Treatment of Hypertension Resulting from Renal Artery Stenosis. George C. Morris, Jr., M.D., Michael E. De Bakey, M.D., John W. Overstreet, M.D., and Russell Scott, Jr., M.D. ... 745
Peter Bryce. Emmett B. Carmichael ... 750
Book Reviews ... 756

No. 12, December 1960

Volvulus of the Stomach. Thomas R. Giblin, M.D., and J. D. Martin, Jr., M.D. ... 759
Spontaneous Rupture of the Liver Associated with Pregnancy: Case Report and Review of the Literature. L. K. Moss, M.D., and J. C. Hudgens, Jr., M.D. ... 763
Hypothermic Circulation with a Temperature Regulating Pump Oxygenator. Allen Greer, M.D., John M. Carey, M.D., and Nazih Zuhdi, M.D. ... 770
An Improvement in the Technique of Cholecystectomy. Carl Bryant Schutz, M.D. ... 773
Spontaneous Rupture of the Spleen in Infectious Mononucleosis. John N. McClure, Jr., M.D., and Ralph A. Huie, Jr., M.D. ... 776
Is Cancer of the Rectum Worthy of the Surgeon's Efforts? Mell B. Welborn, M.D. ... 778
The Complications of Surgical Wounds. Frank Howard, M.D., and Merlin K. DuVal, Jr., M.D. ... 781
Herniated Lumbar Disc—A Surgical Emergency. Homer G. McClintock, M.D. ... 785
Malpractice—Negligence or Misfortune. Carl L. Wilson, M.D., Morton C. Wilson, M.D., and Jerome K. Heilbron, LL.B. ... 788
Experiences in Extracorporeal Circulation. Robert S. Cartwright, M.D., William E. Palich, D.V.M., Thomas P. K. Lim, M.D., Ph.D., Robert Proper, M.D., and David P. Cardus, M.D. ... 792

THE AMERICAN SURGEON

©Copyright 1960, The Williams & Wilkins Co.

Vol. 26, No. 1 January, 1960

THE DIFFERENTIAL DIAGNOSIS OF INTESTINAL OBSTRUCTION IN THE NEWBORN

DAVID B. SHELDON, M.D.*

Los Angeles, California

Intestinal obstruction in the newborn, although not rare, occurs infrequently enough to present a perplexing problem to the physician encountering this difficulty. However, at no other time in the life span is there a greater need for a rapid and accurate diagnosis. The progression of the pathologic physiology due to intestinal obstruction bears an inverse relationship to the size of the patient. Thus, in the newborn infant the course of intestinal obstruction is accelerated, and irreversible complications may develop in the matter of a few hours. Early diagnosis and treatment are imperative.

Intestinal obstruction in the newborn is almost always due to one of several congenital anomalies.[20] Modern techniques and understanding of pediatric anesthesia, fluid replacement and postoperative pediatric care dictate that the majority of these patients should survive the surgery necessary to correct their anomaly. Nevertheless, some deaths in these infants are still attributable to delay in making the diagnosis and carrying out the necessary treatment. The purpose of this paper is to emphasize the early diagnostic features of the common forms of congenital intestinal obstruction encountered in the newborn.

GENERAL CONSIDERATIONS

Polyhydramnios is frequently associated with the births of infants who later prove to have intestinal obstruction. This excess of amniotic fluid is particularly noticeable in cases of esophageal atresia, complete duodenal obstruction, or a high small bowel atresia. The presence of polyhydramnios at the time of birth should immediately alert the obstetrician to the possibility of a congenital obstruction of the intestinal tract.

Prematurity is frequently associated with congenital intestinal obstructions, particularly esophageal atresia. The degree of prematurity bears a direct relationship to the survival rate; however, since the patient has nothing to lose if untreated, prematurity should not prevent the indicated surgery. Certainly the majority of premature infants are not otherwise abnormal, but the presence of prematurity should automatically raise the question of the possible existence of a congenital obstruction of the intestinal tract or of some other hidden congenital anomaly.

Multiple congenital anomalies occur much more frequently than statisticaly expected. Thus the discovery of one congenital anomaly should immediately alert the attending physician to the possible existence of other hidden abnormalities. Esophageal atresia, congenital heart disease, imperforate anus and duodenal obstruction may be associated with one another. Although the presence of more than one serious congenital abnormality automatically lowers the ultimate survival rate, babies frequently survive simultaneous correction of more than one otherwise fatal anomaly. However, only rarely will an infant survive a series of corrections if after the correction of one anomaly, there is postoperative deterioration while an unrecognized second anomaly becomes manifest, and then a second operation is required.

Vomiting is the most prominent and constant symptom of intestinal obstruction. Occasional regurgitation or vomiting is not infrequent in the normal newborn infant, but persistent and pro-

* Assistant Clinical Professor of Surgery, U. C. L. A. Medical School.

gressive vomiting demands immediate investigation. Vomiting of bile, except in the premature, is virtually pathognomonic of organic intestinal obstruction. Excessive salivation and quantitative regurgitation of all feedings are the classical characteristics of an esophageal atresia. Repeated projectile vomiting of gastric contents suggests duodenal obstruction. The onset of vomiting in infants with obstructions lower than the duodenum may be delayed, and the character of the vomitus soon changes indicating a lower small bowel or colonic obstruction. In cases of imperforate anus or congenital megacolon, vomiting does not give as early a diagnostic clue as do distention and obstipation. In general, once vomiting has started it becomes progressively more frequent. The character of the vomitus gives a valuable clue to the level of the obstruction.

Distention as a diagnostic sign depends on the location of the obstruction. The child with esophageal atresia presents with no distention. The child with duodenal obstruction shows only intermittent upper abdominal and epigastric distention consisting of the stomach and sometimes a hugely dilated proximal loop of duodenum. This distention disappears with vomiting. The epigastric fullness of the distended stomach may display visible peristaltic waves familiar at a somewhat later age in cases of congenital hypertrophic pyloric stenosis. Generalized abdominal distention regularly appears, is persistent and progressive in cases of intestinal obstruction lower in the bowel than the duodenum. Such distention may precede the onset of vomiting. The presence of marked distention embarrasses the infant's circulation and respiratory physiology and complicates the necessary surgery. To avoid these serious problems and to prevent further distention from accumulating, gentle gastric suction through a properly placed and frequently irrigated tube is necessary as soon as the probability of intestinal obstruction is determined. Because of the small caliber tube which can be used in an infant, almost constant attention to the management of this tube is necessary.

Obstipation from birth is usually associated with an imperforate anus. An entirely normal appearing meconium stool shortly after birth may erroneously remove the possibility of intestinal obstruction from the mind of the attending physician. The higher in the intestinal tract that an obstruction is located, the greater the chance that the first bowel movement may appear normal. On the other hand, completely normal newborns may show considerable delay before passing their first stool. Usually, however, an infant with intestinal obstruction will show absent or scanty meconium and what stools do appear will never assume the normal milk-curd characteristics. Complete obstipation is eventually present in cases of complete intestinal obstruction. One should not wait for this finding to make the diagnosis because frequently by the time it is clear that true obstipation is present, the infant has long since developed serious dehydration, distention and the other fatal and irreversible associates of intestinal obstruction.

The meconium produced in cases of meconium ileus is characteristic of that condition and may often be recognized by the physician when he does his initial rectal examination by its thick, puttylike, tenacious character. Normal meconium contains the squamous epithelial cells of the vernix caseosa. The absence of these cells, as shown by staining a sample of meconium from the center of the stool with gentian violet, washing, then decolorizing with acidified alcohol (Farber's test),[3] is presumptive evidence of complete intestinal obstruction. Contamination of the specimen with the vernix caseosa near the anus will give a false interpretation.

Partial Obstruction

Partial obstruction may be caused by nearly all of the congenital anomalies which produce intestinal obstruction. Stenosis of all degrees occurs as well as atresia. Malrotation of the colon with duodenal compression may only partially obstruct the bowel and annular pancreas may not completely constrict the duodenum. Meconium ileus varies in severity as does congenital megacolon, and frequently fistulas form adequate escape valves in cases of intestinal obstruction due to an imperforate anus. Some of the most difficult and puzzling diagnostic problems are presented by these partial occlusions of the bowel. There is a direct relationship between the degree of the occlusion, the acuteness of the symptomatology and the immediate danger to the life of the infant. Partial occlusions are most often problems related to other than the neonatal period, symptoms becoming manifest as the baby's diet begins to include solids. Despite the difficulty encountered in securing a diagnosis, these older children rarely present the emergency seen in the complete obstructions, and they are

usually in a more stabilized physiologic condition and often better able to withstand operation than is the newborn infant.

X-ray studies are chief among the aids in making the diagnosis of intestinal obstruction in the newborn and determining its location. Roentgenograms, however, should not be used as a substitute for careful physical examination and appropriate brain work. Plain films of a normal infant only a few hours old will show gas distributed along the intestinal tract. It is important to look for gas in the rectum which is frequently present only a few hours after birth. The absence of these gas shadows, abnormal dilation of the intestine and the presence of fluid levels are pathologic findings.

When the diagnosis of obstruction is in doubt, as it often is early in the course of the disease, invariably there is a temptation to outline and define a blocked bowel by giving barium compound by mouth. It is true that this barium will frequently clinch the diagnosis, but it is equally true that it may severely complicate the subsequent course of the infant. In cases of esophageal atresia, barium introduced into the proximal esophageal pouch will spill over into the respiratory tree and create a severe irritative reaction. In atresias of the duodenum and small bowel, barium from above is a distinct liability. Postoperatively, a minute plug of inspissated or flocculated barium can effectively block the delicate anastomosis that connects the dilated proximal bowel and the distal, tiny, underdeveloped thread of intestine found beyond the atresia. Similarly, barium serves to further obstruct the infant with meconium ileus. If contrast media are necessary, iodized oil (Lipiodol) should be used since it is not as irritating as barium nedia and retains its liquid character.

However, barium studies from below in the form of a barium enema are very helpful in the handling of infants with neonatal intestinal obstructions. A barium enema will give the diagnosis of a congenital megacolon which may masquerade as a low ileal obstruction. A preoperative barium enema will also rule out the possibility of a second atresia or obstruction in the colon. These atresias are difficult to find at the operating table. A barium enema may also give the clue to a malrotation of the colon which is causing duodenal obstruction. This is important because of the frequency with which such a malrotation is associated with volvulus and strangulation of the midgut. The plain film of the baby may also show an abnormal configuration of the cardiovascular shadow suggesting the possibility of an associated congenital heart malformation.

The *pre- and postoperative care* of infants who have congenital intestinal obstruction is equally as important to their ultimate survival as the operative procedure. This subject is extensive and complicated and will not be covered here other than to mention a few of the basic facts.

A newborn infant is in better shape physiologically to withstand surgery within 48 hours of birth than he is 3 or 4 days later. As these golden hours pass, atelectasis and aspiration pneumonia occur, and distention, vomiting, dehydration and electrolyte imbalance become established. Survival rates indicate that it is wiser to treat established dehydration and electrolyte imbalance at least to the point where the infant begins to urinate spontaneously before surgery is undertaken. However, a newborn infant has but little tolerance to overhydration. The cardiovascular and renal systems are not fully mature at birth and thus do not allow as wide a latitude as permissible in the fluid therapy of adults. Pulmonary edema from overhydration is a constant danger. Blood lost during surgery must be promptly and carefully replaced.

A cool, oxygen rich, highly humidified atmosphere helps immeasurably to keep the infant's pulmonary secretions liquid and in such a state that they can be cleared from the tracheobronchial tree by coughing or suction. Chemotherapy is indicated to combat and prevent pulmonary infections. Proper suction should be maintained preoperatively to prevent aspiration of fluid or distention. Postoperatively, gastric suction is maintained until adequate audible peristalsis indicates that the gastrointestinal tract is ready to accept secretions and liquids from above.

The *anesthesia* administered to these tiny patients is also equally as important to their survival as any other aspect of their treatment. The help of a skillful and experienced anesthesiologist is mandatory.

SPECIFIC CONSIDERATIONS

For practical purposes, the diagnosis of intestinal obstruction in the newborn may be divided into four syndromes based on the four common locations of obstruction. Each region has its own symptomatology. These locations are the esopha-

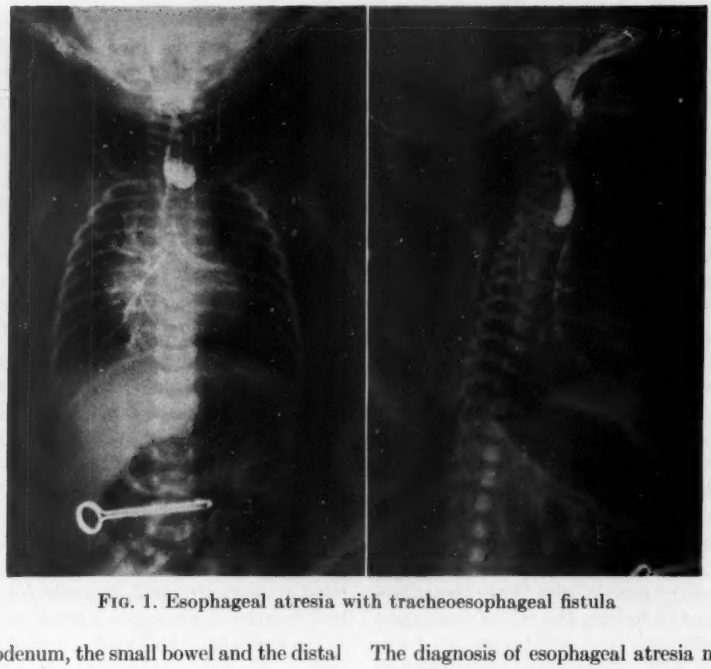

FIG. 1. Esophageal atresia with tracheoesophageal fistula

gus, the duodenum, the small bowel and the distal colon.

Esophageal Obstructions

Esophageal atresia is the cause of obstruction in this region. It is usually associated with a tracheoesophageal fistula and is one of the more common types of intestinal obstruction occurring in the newborn. There are several anatomical variations of this malformation, but over 90 per cent of the cases will have a blind proximal esophageal pouch and a fistula between the posterior aspect of the tracheal bifurcation and the distal esophagus.[12,13]

Although the presence of this abnormality requires major surgery in a newborn infant, the majority of these cases should survive if diagnosed and treated before the development of the pulmonary complications which are invariably associated with this problem. Excessive salivation, coughing, respiratory distress during attempts at feeding and a quantitative regurgitation of all food offered are the early symptoms of this condition.[22] The respiratory symptomatology such as choking, coughing, transitory cyanosis, and the development of a progressive respiratory infection soon becomes prominent as the infant continues to aspirate food and salivary secretions.

The diagnosis of esophageal atresia may be confirmed by attempting to pass a soft rubber catheter (#10–12 F.) into the stomach. This catheter will meet an obstruction at the end of the blind proximal esophageal pouch. X-ray confirmation of the diagnosis may be obtained by instilling a small amount (1 or 2 cc.) of iodized oil (not barium) into the proximal esophageal pouch using the catheter (fig. 1). This radiopaque oil will outline the blind proximal esophageal pouch and demonstrate the lack of communication with the distal gastrointestinal tract. These films also demonstrate the tendency of these infants to aspirate into their tracheobronchial tree the fluids which have pooled in their esophagus. Aspiration pneumonia and atelectasis particularly of the right upper lobe are encountered in all but the earliest cases. Heart size and configuration may give a clue to an all too commonly coexisting congenital cardiac anomaly. Gas bubbles seen in the stomach and intestinal tract demonstrate the existence of the communication between the trachea and the distal esophagus and should be scrutinized for any possible lead to an additional obstruction lower in the gastrointestinal tract. Barium compound should not be used in making these x-ray studies since, when it is aspirated into the tracheobronchial tree, it proves to be irritat-

ing and will further complicate the already precarious pulmonary situation by flocculating and obstructing the tiny respiratory passages.

Esophageal atresia is associated with one or more additional severe congenital anomalies in almost one-third of the cases, and about one-fifth of these babies will be premature. Essentials of treatment of this condition consist first of the re-establishment of the fluid and electrolyte balance. During this period constant oropharyngeal suction prevents further aspiration of salivary secretions. The infant is placed in a warm, moist, oxygen rich atmosphere. Chemotherapy is begun to combat the respiratory infection which almost invariably exists. The surgical approach to the esophageal region may be either transpleural or retropleural. Once exposure has been achieved, the distal esophagus is dissected free from the posterior aspect of the trachea, and the fistula is securely closed. The blind proximal pouch is then dissected free at the apex of the chest, and an appropriate sized hole is cut in the tip of this pouch. Frequently, the tip of a tube passed from above by the anesthetist will help in the initial identification of the proximal esophageal pouch. The cut end of the distal esophagus is dilated to match the hole cut in the tip of the proximal esophagus, and a two-layer anastomosis can usually be performed without undue tension between these two structures.[7, 8] Occasionally mobilization of the stomach is necessary to allow an anastomosis without tension.[14, 15] The mediastinum and chest are then closed. Often it is found to be advantageous to make a gastrostomy with which to feed the infant while the esophageal anastomosis is healing. The most important aspect of the postoperative care is continuous strict attention to the prevention of further aspiration and pneumonia. A heavily humidified atmosphere helps greatly in achieving this objective. Later dilations of the esophageal anastomosis are sometimes necessary in these children. In the cases that do not survive the correction of this anomaly it is often the respiratory complications which cause their death.

Duodenal Obstructions

There are three different anomalies which cause duodenal obstruction. The symptoms from any of these causes of obstruction may be similar, and it is often impossible to distinguish between them. However, they all require exploration and surgical alleviation. Their order of occurrence is malrotation of the colon with compression and obstruction of the second portion of the duodenum by a peritoneal band, duodenal atresia and annular pancreas encircling and compressing the duodenum at the level of the ampulla of Vater.[4] Malrotation of the colon and annular pancreas may produce only partial obstruction. A partially obstructing stenosis of the duodenum may occur instead of an atresia. Thus, the symptomatology may be vague and unclear and not become manifest until later in the life of the child. However, frequently all three of these anomalies produce complete or nearly complete duodenal obstruction.

Early and progressive vomiting is the most prominent symptom. Vomitus usually contains bile; however, in any of these three conditions it may not. The atresia may occur above the ampulla of Vater, the obstructing band in the cases of malrotation may cross the duodenum above the ampulla of Vater and the anatomical relationships in cases of annular pancreas may be such that bile does not regurgitate into the stomach. The majority of these cases, however, do exhibit the vomiting of bile because most atresias are below the level of the ampulla and most malrotations obstruct the second portion of the duodenum below the outlet of the pancreatic and biliary ducts. The vomiting is progressive and persistent and frequently projectile in character. Distention is limited to the upper abdomen, and is intermittent in that when the stomach is emptied by vomiting, the distention disappears. X-ray examination (fig. 2) will show a dilated stomach unless this has just been emptied by vomiting or suction, and there will usually be evident a gas bubble in the dilated loop of duodenum proximal to the obstruction. This shadow is the clue to duodenal obstruction. With complete obstruction the remainder of the abdomen presents a "ground glass" uniform appearance without evidence of gas bubbles or bowel pattern. One may see small amounts (much less than normal) of gas scattered throughout the remaining portion of the intestinal tract in partial duodenal obstruction. This is quite common in both annular pancreas and malrotation of the colon with compression of the second portion of the duodenum.

The introduction of barium to outline more clearly the obstructed region should not be necessary. Air forms a good contrast media in most

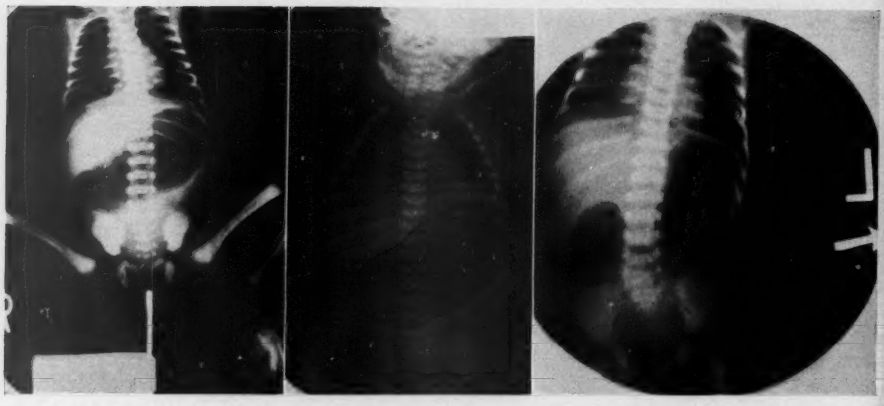

FIG. 2. Duodenal obstruction in the newborn. *Left*, duodenal atresia; *center*, malrotation of the colon with compression of the duodenum; and *right*, annular pancreas.

cases and is not associated with the risk of postoperative obstruction at the anastomosis or in the tiny underdeveloped distal small bowel by flocculated barium. The diagnosis of duodenal obstruction can usually be made on the basis of a flat film without the use of contrast media. However, if contrast media is necessary, it is better to use liquid iodized oil than barium compounds. Once the basic diagnosis of duodenal obstruction has been established, further differential becomes academic because all three of these congenital anomalies require operative treatment as soon as the infant's condition permits. However, if there is to be a delay before surgery for rehydration of an infant with duodenal obstruction, it is important to perform a barium enema which will indicate the presence of a malrotated colon. This must be done because in the cases of malrotation of the colon there is an associated anomalous attachment of the entire midgut mesentery.[5] This consists of a single point of fixation at the origin of the superior mesenteric artery and invites volvulus of the entire small bowel. This volvulus occurs frequently, and if allowed to persist will fatally strangulate the blood supply to the intestine. Infants with an unrecognized volvulus will show the additional symptoms of distention, blood in the stool and finally peritonitis as the bowel becomes necrotic from strangulation and perforates. It is just the child who seems to demand a delay before operation because of toxicity and dehydration who has a volvulus and strangulation of the small bowel blood supply and whose operation cannot wait.

Of course, the surgical technique for correction of each of these anomalies is peculiar to their individual differences. The malrotations are best handled by the method of Ladd[9, 10] which is reduction of the volvulus if it exists and simple division of the peritoneal band extending between the right lateral abdominal wall and the improperly placed cecum. It is this band which compresses and obstructs the second portion of the duodenum. The entire colon is placed in the left side of the abdomen, the duodenum is free. Ordinarily in early cases it is permissible to perform an incidental appendectomy to prevent a later catastrophe from failure to diagnose an appendicitis due to the localization of symptoms in the left upper quadrant.

The atresias of the duodenum present a difficult problem and a somewhat lower salvage rate. The symptoms and signs are the same as for complete duodenal obstruction due to any cause. The difficulty is largely due to the fact that the bowel distal to the atresia has not been dilated to normal size during intrauterine life by having any material passing through it and is extremely small, underdeveloped, and presents only a very tiny lumen, while the duodenum proximal to the atresia is hugely dilated. This discrepancy makes for a difficult anastomosis. With care some dilation of the distal small bowel can be achieved and an anastomosis bypassing the atretic segment of duodenum can be achieved either with the stomach or proximal duodenal loop. This jejunal-duodenal or jejunal-gastric anastomosis may be either retro- or antecolic. Since atresias are fre-

quently multiple, care must be taken to establish that the entire distal bowel is patent. A preoperative barium enema is a much surer way of establishing the patency of the colon than palpation at the operating table. Postoperative care in these patients requires the utmost of patience and persistence, and success depends upon the assumption by the underdeveloped distal bowel of normal peristaltic functions before depletion and malnutrition overtake the infant.

The diagnosis of an annular pancreas frequently presents a difficult problem.[16, 18] Jaundice from involvement of the biliary ductal system may be an added feature. Annular pancreas often does not cause a complete obstruction to the duodenum. Technically, the bypassing anastomosis needed to overcome this anomaly is often easier because the distal bowel is not the tiny underdeveloped caliber seen in the complete atresias. This bowel can be anastomosed to the dilated proximal duodenal loop or to the stomach. It is not proper to attempt division of either the anterior or posterior encircling arm of the pancreas since it is impossible to tell which arm contains the ductal system. Pancreatitis may follow such a division and the embryology of this malformation may be such that even with division of the pancreatic segment one finds that the duodenum is fibrotic and stenosed in this region and does not expand to a full lumen.

Small Bowel Obstructions

Obstructions in the small bowel distal to the duodenal region are usually due to intestinal atresia or meconium ileus.[11] Cases have been recorded of small bowel obstructions at birth caused by congenital peritoneal bands, incarcerated hernias, or duplications of the intestinal tract. However, these causes are very rare.

Small bowel atresia is not common, and it is often difficult to diagnose. This difficulty may lead to a delay which results in a severe deterioration of the baby's condition before operation is undertaken. The atretic segment of bowel may be short and blocked only by a diaphragm of tissue or considerable lengths of the small bowel may be an atretic cord without a lumen. Atresias occur most frequently in the duodenum and the terminal ileal region.[1]

The symptoms will be those of vomiting of gastric contents shortly after birth, then bile containing fluid and finally thin feculent material. The degree of distention which develops depends upon the level of the atresia. In lower small bowel atresias, huge distention and bloating may be seen. The signs and symptoms of dehydration and toxicity become progressively superimposed upon the symptoms of intestinal obstruction. These are listlessness, fever, rapid and weak pulse, anuria and weakness. X-ray films show a generally dilated intestinal tract proximal to the obstruction and no gas shadows in the rectal region (fig. 3). The bowel just proximal to the obstruction may become hugely dilated and finally perforate. No contrast media is necessary to make this diagnosis and should any barium be given from above it may effectively block any anastomosis subsequently achieved. Early in the course of lower small bowel obstructions, peristaltic sounds may give a diagnostic clue by their high pitched rush-like character.

These infants usually need a period of fluid and electrolyte replacement before surgical therapy is undertaken and, during this time, constant suction on a nasogastric tube must be maintained in an effort to prevent any further distention from occurring. Again, the problem at operation is to achieve a satisfactory anastomosis between the greatly dilated segment of bowel proximal to the atresia and the tiny underdeveloped bowel distal to the block. Side to side or oblique anastomoses give the best opportunity for satisfactory function. Following surgery, considerable patience and care are necessary including the administration of parenteral fluids and nutrition before the undilated distal bowel begins to accept thin fluid through the anastomosis from above. The overstretched atonic proximal bowel requires a similar period before it picks up its normal tone and resumes peristalsis.

Meconium ileus causing intestinal obstruction in the newborn is an extremely difficult condition to treat successfully. Meconium ileus represents only one aspect of a widespread generalized disease characterized by failure of the mucus secreting glands of the respiratory and gastrointestinal systems to function properly as well as the failure of the pancreas to form its digestive enzymes.[2] Symptomatologically, meconium ileus may be difficult to distinguish from ileal atresia or megacolon in the newborn (Hirschsprung's disease). However, examination of the meconium in infants so afflicted gives the clue by its peculiarly thick, tenacious character. It is plugs of this dry sticky meconium which block the bowel, usually in the region of the low ileum. Symptoms are vomiting,

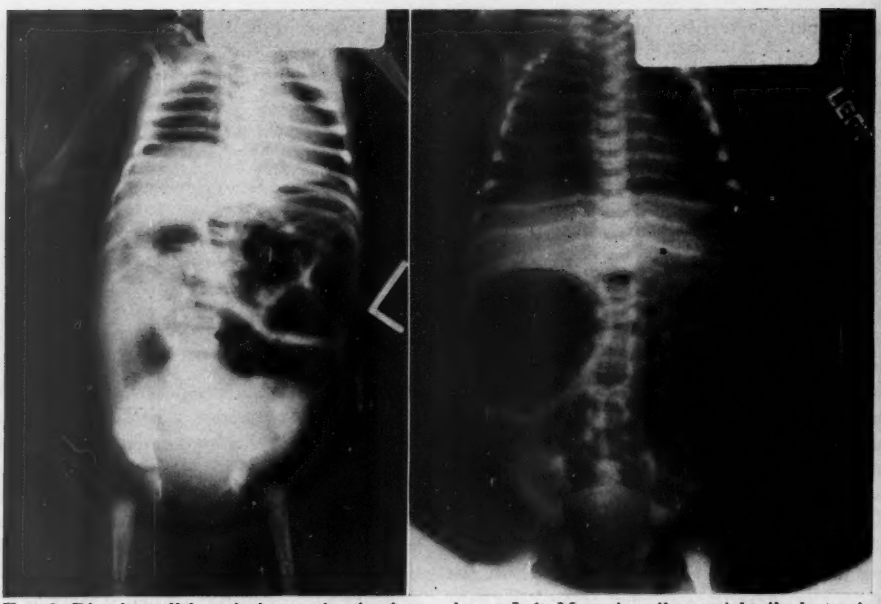

FIG. 3. Distal small bowel obstruction in the newborn. *Left*, Meconium ileus; *right*, ileal atresia

vomiting of bile, increasing distention and obstipation. Pulmonary infections are frequent and large inspissated masses of meconium filled bowel can sometimes be palpated within the abdominal cavity.

Additional confirmation of the diagnosis of meconium ileus can be obtained by laboratory demonstration of the absence of pancreatic enzymes in the infant. A familial tendency for meconium ileus may be noted.

The x-ray picture of the infant with intestinal obstruction due to meconium ileus may show a peculiar coarse, bubblelike appearance in some of the dilated loops of bowel where meconium and gases have intermixed (Neuhauser's sign).

Many methods, both surgical and nonoperative, have been tried in the management of this disease, and none have been found to be very satisfactory. Meconium ileus frequently causes a less than complete obstruction. These cases are best treated in a conservative manner with enemas, gastric lavage and attempts to introduce exogenous pancreatic enzymes from above in the hope that they will come into contact with the obstructing plug and dissolve it. If too much time is spent on these conservative measures without success, the baby's condition may deteriorate beyond where he has any realistic chance of surviving a surgical attempt at removing the block. Occasionally meconium impaction will cause gangrene and bowel perforation. Signs and symptoms of this catastrophe should be carefully watched for. Probably the best chance for the infant with complete bowel obstruction due to meconium ileus rests with early exploration and resection of the obstructed segment of bowel and an oblique anastomosis in continuity. In meconium ileus the distal segment of bowel is usually not the very tiny underdeveloped caliber as found in the atresias and anastomosis is not so difficult.

Unfortunately many of these patients who survive the intestinal obstruction of meconium ileus die before adult life from the pulmonary complications of generalized mucoviscidosis.

Obstruction of the Colon

Imperforate anus and Hirschsprung's disease are the most frequent causes of obstruction to the large bowel of the newborn.

Hirschsprung's disease in the newborn, which is caused by a failure in the development of the parasympathetic innervation of a segment of distal colon,[23] may mimic a low ileal obstruction. Occasionally symptoms may develop shortly after birth.[17] These symptoms are obstipation, distention and vomiting, progressing to feculent vomiting. Plain x-ray films of the abdomen

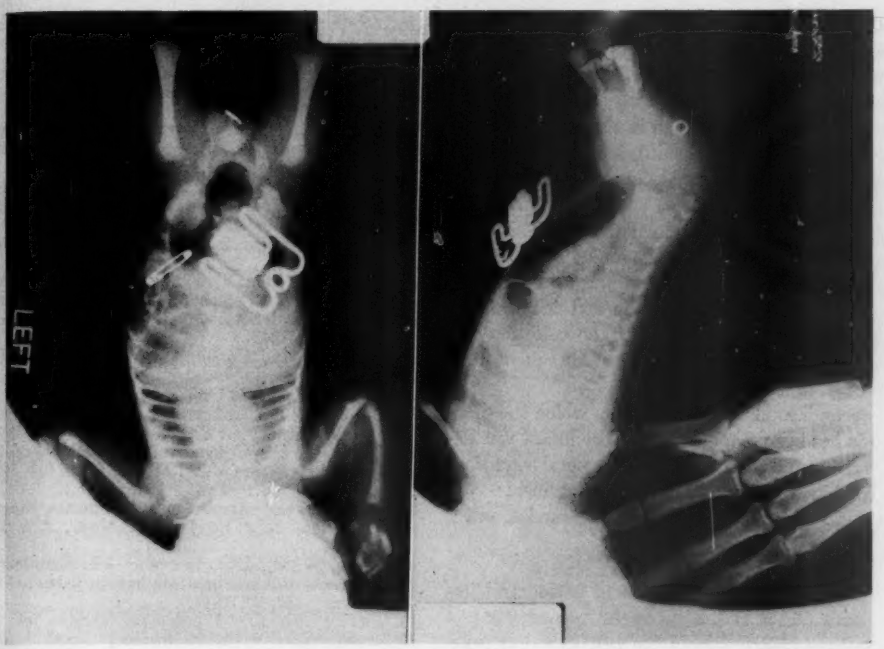

FIG. 4. Imperforate anus

do not always distinguish this condition from a low ileal atresia. Dilated bowel with fluid levels may be present, and it is not uniformly possible to tell whether the colon is involved in this dilation. A barium enema will prevent a needless and sometimes fatal operation since Hirschsprung's disease in the newborn can usually be treated conservatively with enemas. The diagnosis is confirmed by rectal biopsy as described by Ovar Swenson. Later when the child has reached a suitable age, definitive surgical therapy can be undertaken with greater safety.

Imperforate anus is probably the most frequent congenital anomaly requiring surgery in the newborn. It occurs in several forms, varying from a mild anal stenosis to a blind rectal pouch ending high in the pelvis. The majority of cases are of the latter type. Three-fourths of the infants will have a fistula connecting this blind rectal pouch to the bladder, urethra or perineum in males, or to the vagina or perineum in females. Careful rectal examination of all newborn infants is routine and generally discloses the presence of an imperforate anus. Occasionally this examination is missed or the atresia may be located above the reach of the fingertip.

In these cases the symptoms of intestinal obstruction develop shortly after birth, first obstipation, then abdominal distention and finally vomiting. Dehydration and the manifestations of the toxic effects of intestinal obstruction follow soon afterward. If there is an associated fistula of sufficient size, the infant may pass meconium and gas through the urinary tract or from the vagina. Occasionally a perineal fistula is so large as to permit bowel function so nearly normal that the true nature of the rectal abnormality is not discovered until the patient is older.

X-ray studies are helpful in determining the level of the atresia (fig. 4). If the child is held inverted, a plain abdominal film will show gas rising to the distal end of the colon and by marking the anal dimple with lead the distance between the blind pouch and the surface can be determined.[24] For these studies to be significant the child must have passed enough gas through the length of the intestinal tract to outline the blind end of the colon.

The treatment chosen for this condition depends upon the anatomical type of the anomaly. In cases of anal stenosis, repeated and gradually increasing dilation solves the problem. If there is

only a thin membrane obstructing the anus, a simple perineal perforation of this membrane followed by serial dilations is all that is necessary. In the most common type in which the rectum ends in a blind pouch high in the pelvis (an inch or more from the anal skin), two types of treatment must be considered. First, a simple colostomy may be performed, and the definitive reconstruction of the anal canal deferred. This is the procedure of choice when the infant is premature, or when its condition is precarious due to the intestinal obstruction, distention and dehydration. Care must be taken in doing the colostomy to leave ample distal bowel for the subsequent pull-through procedure.

Second, in ideal cases, those in which the diagnosis has been made early and the infant was delivered at full term and in good general condition, a combined single stage abdominal-perineal reconstruction of the rectal canal can be carried out.[19]

SUMMARY

Early diagnosis and treatment is the most important factor in the successful management of intestinal obstruction in the newborn. For practical purposes the diagnosis can be divided into four syndromes which correspond to the four locations in which these obstructions are likely to occur; namely, the esophagus, the duodenal region, the small bowel and the colon. Vomiting of bile is almost pathognomonic for intestinal obstruction except in the premature infant. Distention and obstipation are also important symptoms and may precede the vomiting depending on the location of the obstruction. Plain x-ray films usually confirm the diagnosis. Barium should not be given from above but a preoperative barium enema is a necessity. The anesthesia, pre- and postoperative care are equally as important as the surgery in the care of these infants.

Dept. of Surgery
U. C. L. A. Medical Center
Los Angeles 24, California

REFERENCES

1. Biggs, A. D., and Pontius, G. V.: Intestinal obstruction in the newborn. J. Pediat., *30:* 306, 1947.
2. Farber, S.: The relation of pancreatic achylia to meconium ileus. J. Pediat., *24:* 387, 1944.
3. Farber, S.: Congenital atresia of the alimentary tract: diagnosis by microscopic examination of meconium. J. A. M. A., *100:* 1753, 1933.
4. Fox, P. F.: Duodenal obstruction in infants and children. A. M. A. Arch. Surg., *67:* 475, 1953.
5. Gardner, C. E., Jr.: The surgical significance of anomalies of intestinal rotation. Ann. Surg., *131:* 879, 1950.
6. Gross, R. E.: *The Surgery of Infancy and Childhood.* W. B. Saunders Company, Philadelphia, 1953.
7. Haight, C.: Congenital atresia of the esophagus with tracheo-esophageal fistula. Reconstruction of esophageal continuity by primary anastomosis. Ann. Surg., *120:* 623, 1944.
8. Haight, C., and Towsley, H. A.: Congenital atresia of the esophagus with tracheo-esophageal fistula. Surg., Gynec. & Obst., *76:* 672, 1943.
9. Ladd, W. E.: Congenital obstruction of the duodenum in children. New England J. Med., *206:* 277, 1932.
10. Ladd, W. E.: Congenital duodenal obstruction. Surgery, *1:* 878, 1937.
11. Ladd, W. E.: Congenital obstruction of the small intestine. J. A. M. A., *101:* 1453, 1933.
12. Ladd, W. E.: The surgical treatment of esophageal atresia and tracheoesophageal fistulas. New England J. Med., *230:* 625, 1944.
13. Ladd, W. E., and Swenson, O.: Esophageal atresia and tracheo-esophageal fistula. Ann. Surg., *125:* 23, 1947.
14. Leven, N. L.: Congenital atresia of the esophagus with tracheoesophageal fistula. J. Thoracic Surg., *10:* 648, 1940–1941.
15. Longmire, W. P., Jr.: Congenital atresia and tracheo-esophageal fistula. Arch. Surg., *55:* 330, 1947.
16. Moore, T. C.: Annular pancreas: review of the literature and report of two cases. Surgery, *33:* 138, 1953.
17. Potts, W. J., Boggs, J. D., and White, H.: Intestinal obstruction in the newborn infant due to agenesis of the myenteric plexus (congenital megacolon). Pediatrics, *10:* 253, 1952.
18. Ravitch, M. M., and Woods, A. C., Jr.: Annular pancreas. Ann. Surg., *132:* 1116, 1950.
19. Rhoads, J. E., Pipes, R. L., and Randall, J. P.: A simultaneous abdominal and perineal approach in operations for imperforate anus with atresia of the rectum and rectosigmoid. Ann. Surg., *127:* 552, 1948.
20. Sheldon, D. B.: Emergency operations in the newborn. California Med., *88:* 221, 1958.
21. Swenson, O.: *Pediatric Surgery,* Appleton-Century-Crofts, Inc., New York, 1958.
22. Swenson, O.: Diagnosis and treatment of atresia of the esophagus and tracheoesophageal fistula. Pediatrics, *1:* 195, 1948.
23. Swenson, O., Rheinlander, H. F., and Diamond, I.: Hirschsprung's disease: a new concept of the etiology. Operative results in thirty-four patients. New England J. Med., *241:* 551, 1949.
24. Wangensteen, O. H., and Rice, C. O.: Imperforate anus—a method of determining the surgical approach. Ann. Surg., *92:* 77, 1930.

THE USE OF HYPOTHERMIA IN SEPTIC SHOCK*

JOHN M. ALLEN, M.D., JAMES T. ESTES, M.D., AND ARLIE R. MANSBERGER, JR., M.D.

Baltimore, Maryland

Peripheral vascular collapse associated with the blood stream invasion of bacteria or their products has been called septic shock. The pathophysiologic mechanisms responsible for this symptom complex are poorly understood even though it is not of infrequent occurrence[3-5] and can be produced experimentally in a variety of ways.[7, 16, 24]

Regardless of the mechanisms involved in the production of septic shock, the syndrome is attended by a high mortality rate. In 1956 Altmeier and Cole[3] reported 93 patients who exhibited evidence of clinical shock in association with a serious infection. All of these patients died. Ezzo and Knight[9] in 1957 reported a 66 per cent mortality in 38 patients treated for septic shock.

At present the treatment of shock associated with infection consists of large doses of an appropriate antibiotic, vasopressor substances and corticoids. Peterson and Krippaehne[25] cite the detrimental effect of hyperthermia and have stressed the importance of lowering the temperature to normothermic levels in this group of patients. Reeves and Lewis[26] noted a definite clinical improvement in critically ill, febrile patients who were cooled to 35 to 36°C. To our knowledge hypothermia has not been utilized heretofore in the treatment of patients with septic shock.

As early as 1798, Currie[5] reported lowering the body temperature of a healthy individual to 83° while keeping him in a brine bath at 40° for a period of 45 min. In 1938 Fay and Henny[11] found that the regional lowering of body temperatures in patients with far advanced malignancy was an effective means of relieving pain. A year later Fay and Smith[12] utilized "prolonged human refrigeration" as a method of controlling pain in patients with generalized carcinomatosis and reported the first detailed observations on the effect of hypothermia in the human being. In 1941 Fay[10] demonstrated the benefit of lowered body temperature on patients with cerebral trauma.

The effects of some poisons have been tested during hypothermia. In 1934 M. L. Tainter[27] found that dinitrophenol injected into pigeons and guinea pigs failed to produce the usual toxic effect if the environmental temperature was lowered to 38 to 42° and that only 25 per cent of the birds so treated died.

Hypothermia has been tried in cases of various infections. Akiyomae[1] innoculated a series of ground squirrels with the virus of lymphopathia venerea. The controls died, whereas squirrels kept at reduced temperatures for 20 days awakened unharmed and without clinical signs of disease.

The successful use of hypothermia in infection depends upon a retardation of bacterial metabolism and growth with a lesser effect upon the bacterial defense mechanisms of the host. Eiseman and co-workers[8] made this conclusion from observations during experiments in which they induced pneumococcal peritonitis although they presented no experimental evidence to support this claim. Balch and associates,[4] on the other hand, were unimpressed with the therapeutic efficacy of lowered body temperatures in experimental peritonitis in rats and concluded that bacterial proliferation and bacterial defense mechanism were both retarded by surface cooling.

Friedman and co-workers[19] have reported that hypothermia protects the antibacterial defense mechanism ordinarily injured by hemorrhagic shock and showed that the animals in shock may cope with a dose of bacteria injected during the hypothermic period that would be fatal to normothermic dogs.

Mansberger and his associates[22] demonstrated a beneficial effect of local hypothermia on wound shock and showed that a delay of definitive debridement in their animals resulted in indefinitive survivals only when local hypothermia was employed in addition to antibiotics and restoration of blood volume. Allen[2] has demonstrated a dramatic blood pressure response

* From the Department of Surgery, University of Maryland School of Medicine, Baltimore, Maryland.

Supported by the Research and Development Division, Office of the Surgeon General, Department of the Army, Washington 25, D. C., Contract No. DA-49-007-MD-674.

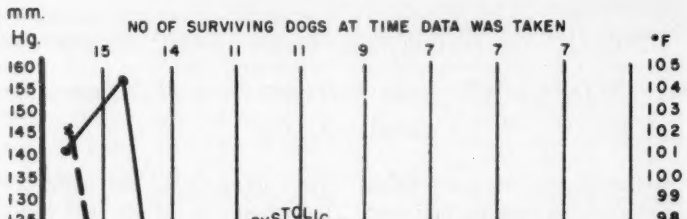

FIG. 1. Systolic blood pressure response to hypothermia in dogs with fecal peritonitis

to surface cooling in dogs in shock from fecal peritonitis (fig. 1).

Fedor and co-workers[14] studied the effect of hypothermia on the bacterial defense mechanisms of dogs. They observed that not only is the leukopenia which is characteristically described in hypothermia transitory in nature and followed by an increase in band neutrophils but also that the leukocytes did not lose their ability to phagocytose organisms either during the phase of cooling or following rewarming. In addition these same authors demonstrated by paper electrophoresis that no change occurred in phagocytic promotion factors of plasma during hypothermia.

Fedor and his colleagues[15] showed that hypothermic dogs were able to clear their blood streams of 99 per cent of 5 to 50 billion injected pathogens within 6 hr. as compared to 100 per cent clearance by normothermic animals during the same period.

Thus, experimental evidence to date suggests that there is in fact a retardation of bacterial growth and metabolism under conditions of lowered body temperature and a much less significant depression of bacterial defense mechanisms under the same conditions. These observations plus the reduction in oxygen and metabolic requirements of the body produced by general hypothermia suggest further that its use might be of benefit to patients in septic shock. The following patients in septic shock were treated by hypothermia only when their progressive deterioration was apparent after failure to respond to accepted therapeutic regimens.

CASE REPORTS

*Case 1.** E. S., a 35-year-old man was admitted to the University Hospital on August 14, 1958, with generalized peritonitis from perforation of a duodenal ulcer. The perforation had occurred 20 hr. previously. On admission he was in shock and comatose. Following gastric intubation and intravenous resuscitative therapy, closure of the ulcer was accomplished. During operation, 3000 cc. of a greenish gray, tenacious, foul-smelling purulent fluid was removed from the peritoneal

* Presented at the University of Maryland to a joint meeting of the Sections of Infectious Disease of the Johns Hopkins University School of Medicine and the University of Maryland School of Medicine, October 2, 1958.

cavity. Culture of this material grew out *Neisseria catarrhalis*, α-hemolytic Streptococcus, *Escherichia coli* and a Gram-negative rod of the Hemophilus group.

On the 3rd postoperative day there was sudden increase in temperature to 104°. His subsequent clinical course over the next 5 days was one of progressive deterioration characterized by increasing distension, absence of peristalsis, tachycardia, delirium and a gradual but steady fall in blood pressure in spite of vigorous fluid, electrolyte and antibiotic therapy.

On the 8th hospital day a 2nd laparotomy was performed for drainage of a large pelvic abscess. When the peritoneal cavity was opened, distended cyanotic loops of bowel presented through a large accumulation of purulent fluid. Following drainage of the pelvic abscess and evacuation of peritoneal fluid, the peritoneal cavity was lavaged with saline and a solution of oxytetracycline. Cultures of the abscess cavity revealed a pure growth of *E. coli* which was sensitive only to kanamycin sulfate.

Again temporary improvement in the patient's general condition was interrupted by a shaking chill and elevation of temperature to 104°. Coincident with the temperature elevation, the patient's blood pressure rose briefly to mildly hypertensive levels. This rise was followed by a precipitous fall in blood pressure, cardiac failure with pulmonary edema, and delirium. Electrocardiographic changes were compatible with a toxic myocarditis. In the next 15 hr. an intensive therapeutic regime was carried out. Treatment over this period consisted of rapid digitalization, oxygen per nasal catheter, Hydrocortisone via the intravenous (i.v.) route, i.v. vasopressors and kanamycin sulfate. Except for an initial blood pressure response to i.v. hydrocortisone and vasopressors the patient's clinical condition remained unchanged. An increasing resistance to vasopressors was evinced by a gradual decline in blood pressure (B. P.) in spite of the use of increasing increments of vasopressors. During this period the patient's wound separated down to the posterior rectus sheath and an ileal fistula became evident.

By 2 p.m. on the 11th hospital day the patient appeared terminal. His heart sounds were faint and irregular at 168 beats per min., B.P. was 60/0, and respirations were shallow, 50 per min., corneal reflexes were still present, but the patient responded only to painful stimuli. Urinary output had fallen to 10 cc. per hr. At this point the body temperature was reduced by surface cooling. With the blanket temperature maintained at 40°, the sigmoid temperature fell to 90° over an 8-hr. period. Physical evidence of shivering was absent during the induction period. With this reduction in body temperature, dramatic improvement became evident (figs. 2 and 3). The pulse again became regular, 100 per min., the B.P. stabilized 130/90 mm. Hg, the urinary output increased to 60 cc. per hr., and the sensorium cleared.

For the next 16 days the sigmoid temperature was maintained in the range of 90 to 93°. I.v. kanamycin sulfate therapy was continued during this period. Brief attempts to rewarm the patient on the 3rd, 5th and 11th days of hypothermia were followed by exacerbations of the precooling state including a fall in hourly urinary output.

Rewarming was slowly accomplished between the 28th and 33rd hospital days (16th through the 21st were days of subnormal temperature). During the period of hypothermia several observations were made which are worthy of note:

1. The analgesic effect of surface cooling was evinced by lack of need for any narcotic during this period. Cannulations of peripheral blood vessels were accomplished without local anesthesia and without evidence of pain.

2. Retrograde amnesia in this patient could be dated to the period immediately preceding hypothermia even though the sensorium appeared normal when body temperatures were maintained at 91 to 93°. The patient was unable to recall any events which occurred during refrigeration.

3. On several occasions a clouded sensorium responded to i.v. *l*-arginine administration even though peripheral arterial ammonia levels were within normal limits.

4. Potassium shifts were evinced by a fall in serum potassium below 2 mEq. per L. during the attempts to rapidly rewarm the patient on the 11th day of hypothermia.

5. There was no evidence of wound healing during the period of cooling. Granulation tissue "was conspicuous by its absence."

6. On several occasions (after the 5th day of cooling) the patient drifted into unconsciousness followed by a decrease in B.P. and respiratory rate. During such states it was found that the blood sugar was dangerously low (table 1). On one occasion a value of 5 mg. per cent was obtained, in spite of the daily administration of 10 per cent glucose. These hypoglycemic episodes responded to the intravenous administration of 50 per cent glucose and fructose. Blood sugars of 40 to 50 mg. per cent were well-tolerated.

The patient's wound healed rapidly after rewarming, and by the 40th hospital day the wound was closed except for the fistulous opening.

During the first 31 days of hospitalization, the patient lost 32 lb. of body weight. By the time of discharge from the hospital on the 84th hospital day he had regained 28 lb.

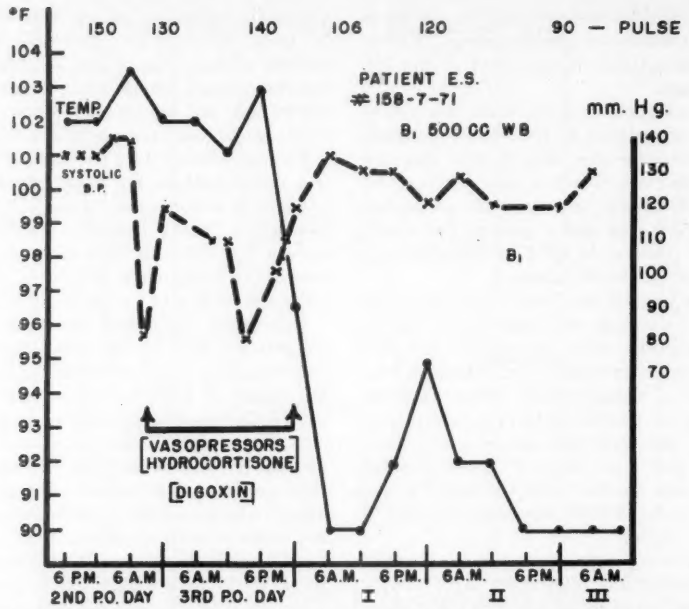

FIG. 2. Septic shock in case 1, patient E. S. Pulse and blood pressure response to supportive measures and to the initial three days of hypothermia (*I, II, III*). Note the increase in pulse and fall in blood pressure with moderate rewarming.

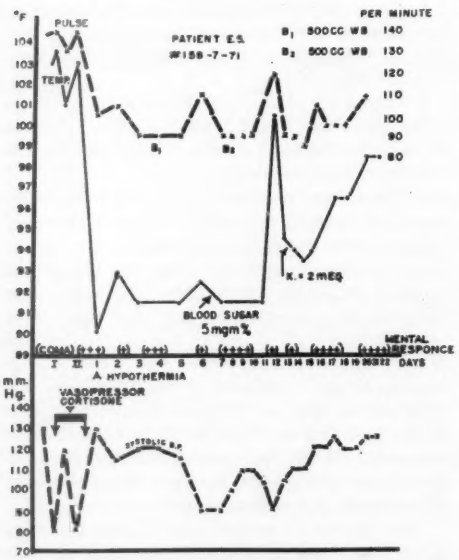

FIG. 3. Septic shock in case 1, patient E. S. Response of pulse and blood pressure during 21 days of hypothermia. Note that homeostasis was obtained with gradual rewarming.

The patient's ileal fistula has subsequently been closed, and he is now gainfully employed as a laborer. A point of interest was the striking lack of intraabdominal adhesions at the time of closure of the ileal fistula.

A battery of studies done 2 to 5 weeks after rewarming which included liver function studies, glucose tolerance, Bromsulphalein (BSP), phenolsulfonphthalein test (PSP) and electrocardiograms were all within normal limits.

Case 2. G. O., a 72-year-old man with a history of chronic pulmonary fibrosis and emphysema, was admitted to the medical service of University Hospital on February 7, 1959, with massive upper gastrointestinal hemorrhage. On admission his B.P. was 90/60 mm. Hg. He was vomiting blood and passing tarry stools. In the next 18 hr. he was given a total of 6 pints of blood. At this point the patient had a cardiac arrest. A cardiac puncture was carried out in bed and 1 cc. of 1:20,000 Adrenalin was administered. Within 30 sec. the heart beat was again audible, and the B.P. was 110/70. The patient continued to hemorrhage. On February 8, 1959, a subtotal gastric resection was accomplished with excision of a bleeding duodenal ulcer. Fifteen hundred cc. of whole blood were given during the operation. On the evening of the

TABLE 1

*Blood studies and other data**

	Precooled Period in Days		Hypothermic Period in Days															Normothermic Period in Days								
	I	II	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25
Average temperature	104.5†	103.5†	90	91	91.5	91.5	92.5	92.5	91.5	91.5	91.5	91.5	91.5	96	94.5	94	93.5	94	95	96	96	97.5	98	98	99	99	
Hemoglobin	16	13	15			15		16	15	15		14		13			13			14			12			12	12
Hematocrit	50	41	45			48		51	47	49				40			43			41			36				
Blood sugar							5/134	46	26	19	80	99	80	58	77	117	79	92		96	90					77	61
Blood urea nitrogen	36	28	27	39		36	33	23	18	18	15	12	13	10	10	12	14			13	15					8	11
CO_2		17				24		19		24	20	22	24	27	33	24	26	26			23					20	22
Cl	118	113		103		100	101	97	107	101	93		98	105	92	88	86			113	100					92	93
Na	143	137	137	145		140	139	128	136	135	134		142	144	141	134	135			147	139					128.9	129
K	5.5	4.7		5.1		4.6	4.9	5.5	5.6	5.0	5.0		4.2	4	2	3.3	3.2			5.3	4.6					4.9	5.1
Total protein						6.3																					7
Whole blood transfusion						500		a.m. 500																			
Blood volume						−1000 cc.		p.m. +850 cc.																			

* Case 1, E. S., Hosp. No. 158-7-71.

† Peak temperatures.

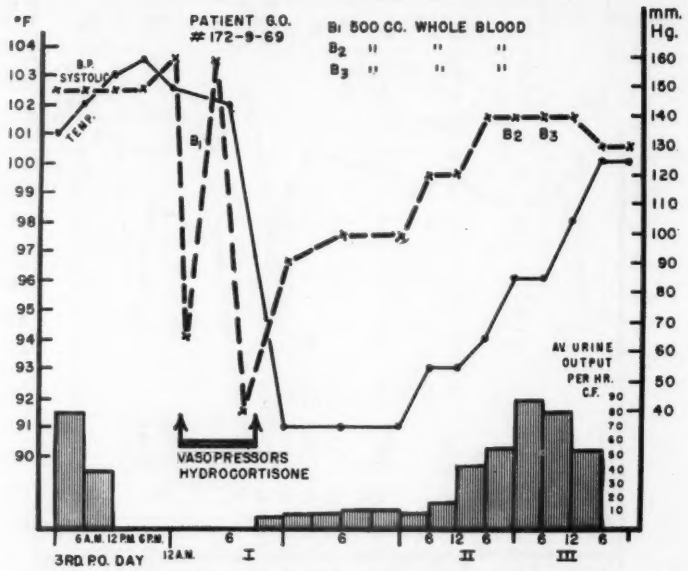

FIG. 4. Septic shock in case 2, patient G. O. Response of blood pressure and urinary output to hypothermia. Note that anuria did not respond during the normotensive period obtained with vasopressors.

1st postoperative day the patient developed tachypnea and cyanosis and at bronchoscopy was found to have a left apical pneumothorax. A thoracotomy tube was placed in the 2nd left intercostal space and connected to an underwater trap drainage system. Within 24 hr. the air leak had sealed, the lung was completely re-expanded, and the thoracotomy tube was removed.

On the 3rd postoperative day he developed a severe shaking chill followed by a temperature elevation to 103.5°. A blood culture taken during the chill later grew out hemolytic *Straphylococcus aureus* (coagulase positive) and *E. coli*. Sputum cultures taken at the time of bronchoscopy grew *Pseudomonas pyocyanea*, *E. coli* and hemolytic *S. aureus* (coagulase positive). Sensitivity studies revealed the organisms to be sensitive to chloramphenicol, and the patient was given 1 gm. of this drug every 8 hr. via the intravenous route. Over the next several hours, the B.P. remained stable at 150/80 mm. Hg, but the hourly urinary output decreased dramatically in spite of an adequate intake. At 1 a.m. on the 4th postoperative day the B.P. was unobtainable, and the patient was unresponsive. He was given 500 cc. of whole blood in spite of the fact that a blood volume done the previous morning had shown an excess of 302 cc. A total failure of B.P. response prompted the use of intravenous vasopressors (Vasoxyl) and hydrocortisone. A temporary rise in B.P. to 80/50 was followed by a gradual decline in spite of the use of increasing amounts of Vasoxyl.

By 8:30 a.m. on the 4th postoperative day, the B.P. had fallen to 40/0. (The oliguria which had started several hours before the precipitous fall in B.P. did not respond to the temporary manometric rise, and the patient became and remained aneuric for a total of 22 hr.) The patient appeared moribund, and hypothermia was induced. His body temperature was reduced from 102° to 93° over a 2 hr. period. Shivering which started at 96° was controlled with a single dose ot chlorpromazine. The temperature was maintained between 91 and 93° over the next 51 hr. Coincident with the hypothermic state, the B.P. rose quickly to 90/50 and more gradually to 140/70 mm. Hg at which point it stabilized. The hourly urinary output which was 10 cc. after the 2nd hour of hypothermia, gradually increased to 60 to 80 cc. per hr. (fig. 4). The patient's sensorium gradually cleared so that he was completely alert and responsive by the 6th hypothermic hour. After 51 hr. of hypothermia, the patient was allowed to rewarm.

During the rewarming phase, the patient had a drop in hemoglobin and was given 1000 cc. of whole blood. Evidence of cardiac failure was noted at this time, and the patient was digitalized. A blood volume determination revealed a total excess of 2250 cc., 963 cc. of which was due to expansion of the red cell mass (table 2).

TABLE 2

*Blood studies and other data**

	Precooled Period in Days			Hypothermic Period in Days								
	I	II	III	1	2	3	4	5	6	7	8	9
Average temperature	101†	102†	103.5†	91	93	96	99	100	101	99	100	100
Hemoglobin	14	13		12	14	11		14	13		10	11
Hematocrit	36	40		39	43	40		44	40		33	32
Blood sugar				89	110	208	133	136	197	136	188	
Blood urea nitrogen	48	66	43	66	70	87	48	34	33	54	60	
CO_2	22	31		28	31	30	37	36	38	27	26	26
Cl		100		90	88	102			100	105	92	100
Na		144		146	134	149			144	142	138	132
K		3.7			4.4	4.9			5.5	4.7	3.8	4.0
Total protein			5.2		5.4							
Whole blood transfusion				250 cc.		1000 cc.						
Blood volume		+302 cc.					+2250 cc.					

* Case 2, G. O., Hosp. No. 172-9-67.

† Peak temperatures.

Blood cultures taken subsequent to rewarming remained sterile. The patient's course over the next several days was relatively uneventful and by the 17th hospital day he was ambulatory and taking an adequate diet.

Case 3. A 55-year-old woman, inmate of a mental institution, underwent an operation at University Hospital on April 11, 1959, for sigmoid colon volvulus of 6 days' duration and perforation of 2 days' duration. Cultures from the peritoneum grew out *E. coli*.

Shortly after the induction of anesthesia the patient's B.P. dropped to 60/0. She subsequently received 1000 cc. of whole blood and intravenous vasopressors which maintained the B.P. at 100/60 for a brief period. At the end of the procedure, however, the B.P. and pulse became inaudible in spite of vasopressors and additional whole blood transfusion.

Hypothermia was induced by surface cooling. During the first 2 hr. of cooling the temperature rose from 99 to 101°, and marked shivering was evident. The shivering was controlled, and a subsequent fall in temperature to 86° was attained within a period of 5 hr. In this interim the B.P. rose to 90/20, the pulse became full and regular at a rate of 90 per min. The patient responded to questions. The urinary output which had been 0 cc. per hr. for 12 hr. increased to 20 cc. per hr. for a period of 6 hr. after which hemoglobinuria developed, and renal output decreased progressively to anuria over the next 16 hr. (fig. 4).

During the next 24 hr. the patient's vital signs were stable. However, by the 2nd day of hypothermia, massive hemolysis became evident in the serum samples taken. Serum hemoglobin on the evening of the 2nd postoperative day was 2 gm. per cent. The B.P. dropped progressively over an 8-hr. period, and the patient expired (fig. 5).

This case is mentioned as an example of initial septic shock which responded to hypothermia but later succumbed to an overwhelming hemolysis which may have been due to blood incompatibility.

Case 4. M. L. M., a 62-year-old female physician with a long history of mitral stenosis and pulmonary insufficiency developed multiple abscesses and pneumonia of the left lung 3 months after a course of irradiation therapy for bronchogenic carcinoma. Cultures of the purulent material obtained from below a left bronchial stenosis grew out *Klebsiella pneumoniae*, *Clostridium perfringens* and *S. aureus*. The *K. pneumoniae* was sensitive to neomycin, polymyxin B-sulfate and kanamycin sulfate.

The patient subsequently received a combination of polymyxin B-sulfate and chloramphenicol for a period of 6 days. During this time she spiked temperatures to 102° each day and deteriorated clinically. There was roentgenologic evidence of progression of the pneumonia and further abscess formation.

Because the right lung was not involved and the infection in the left lung was progressing, a left

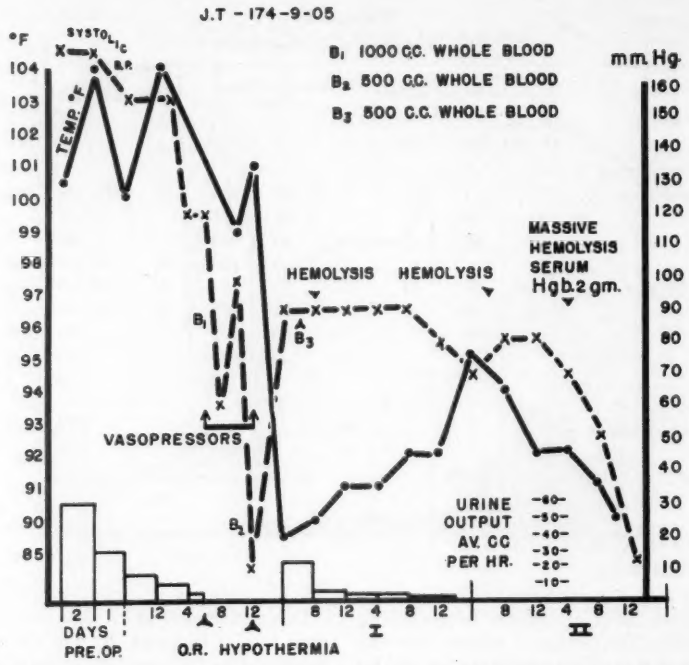

FIG. 5. Septic shock in case 3, patient J. T. Response of blood pressure and urinary output to vasopressor and hydrocortisone, and to hypothermia. Note the absence of response in the face of massive hemolysis.

pneumonectomy was performed. In view of the illness of the patient and anticipating a septic postoperative course, it was decided that the procedure should be done at subnormal temperatures which were to be maintained for an indefinite period.

The patient's temperature was lowered by surface cooling to 93° before the induction of anesthesia. During the operative procedure a large multilocular abscess was inadvertently ruptured, and there was spillage of 150 cc. of thick yellow, foul-smelling pus into the left pleural space. The pneumonectomy was subsequently accomplished without further untoward incident.

After the procedure, the patient's temperature was maintained in the range of 92 to 94° for a period of 2 days, 94 to 95° for a day and 95 to 96°F for an additional 4 days after which she was allowed to rewarm.

The postoperative course was relatively uneventful. By the end of the 1st postoperative day, the pulse ranged from 70 to 80 per min., the blood pressure stabilized at 130/70 mm. Hg, and the respirations ranged from 20 to 30 per min. Cultures of aspirated pleural fluid were negative by the 8th postoperative day, and the antibiotics were discontinued the following day. The period of hospitalization, however, was prolonged because of the numerous thoracentesis which were required to aspirate fluid accumulations in the left pleural space. Repeated cultures of pleural fluid were sterile. She was discharged on the 60th postoperative day.

DISCUSSION

The fact that all patients treated experienced relief of pain corroborates the experience of Fay and Smith,[12] who utilized both hypothermia and local refrigeration for relief of pain in patients with carcinomatosis. These authors also observed and reported retrograde amnesia during the period when their patients' temperatures were 92° or below even though reasoning powers were retained during the same period.

The hypoglycemic episodes which occurred in patient E. S., beginning after the 5th day, were probably in part the result of hepatic glycogen depletion in spite of the daily administration of 10 per cent glucose. Fisher and associates[17] demonstrated a decrease of 63 per

cent in blood sugar values in dogs after 19 hr. of hypothermia. Their animals showed a 4+ qualitative urine sugar throughout the period of hypothermia. Utilizing periodic acid-Schiff staining techniques, Fisher and co-workers[18] were able to demonstrate a depletion of glycogen in peripheral portions of hepatic lobules of dogs sacrificed after 4 to 6 hr. of hypothermia.

Wynn,[28] on the basis of abnormally high glucose tolerance curves in dogs and in one human being, suggested that during induced hypothermia exogenous glucose is metabolized slowly. That glucose utilization is impaired during hypothermia is also suggested by the experimental work of Henneman and associates[20] These authors suggest that the impairment of tissue uptake of glucose may be related either to inhibition of insulin activity or to an inhibition of hexokinase which is "uniquely sensitive to temperatures changes."

The hypokalemia observed in patient E. S. is undoubtedly the result of several factors. During the hypothermic phase of this patient's illness (even though he was maintained throughout this phase by total i.v. alimentation) his ileal fistula continued to discharge moderate amounts of succus entericus. Although urinary excretion of potassium was not measured, it is probable that increased amounts of potassium were lost in the urine during refrigeration.[23] Because the hypokalemic episodes occurred during attempts to rapidly rewarm the patient, it is likely that the dilution factor of "returning plasma" may have been contributing.

The complete lack of attempts at wound repair observed in patient E. S. deserve special comment. Löfström and Zederfeldt[21] found that the tensile strength of incisions in experimental hypothermic rabbits was reduced by as much as 29 per cent. They cited intravascular aggregation of blood cells with accompanying changes in capillary circulation as a cause for retardation of wound healing after induced hypothermia. By infusing low molecular weight dextran they were able to prevent intravascular aggregation of corpuscles during hypothermia and could show no significant decrease of tensile strength in experimental wounds. Biochemical and histochemical alterations of wounds have not been documented under conditions of induced hypothermia. It is certainly possible that the formation of mucopolysaccharides, precollagen and collagen, are impaired, perhaps, because of deficient utilization of glucose. There can be little doubt, however, that the prolonged period of negative nitrogen balance in this patient was a major factor in the failure of his wound to heal during the period of refrigeration.

Patient G. O. went into cardiac failure upon rewarming. The fact that he received whole blood during the depths of hypothermia was undoubtedly a contributing factor. It will be recalled that his blood volume following rewarming revealed an excess of 2250 cc. D'Amato and Hegnauer[6] have demonstrated that the plasma volume of dogs subjected to immersion hypothermia was diminished by 12 per cent without change in the red cell mass. Fedor and Fisher[13] substantiated these findings and showed that the values returned to normal within 24 hr. after rewarming. There is a general agreement among workers in hypothermia that the increase in hematocrit in hypothermia is secondary to decreases in plasma volume with no change in the erythrocyte volume. The reduction in plasma volume has been attributed to capillary leakage or to plasma trapping in small peripheral vessels. The exact mechanism is unknown. Regardless of cause, it would seem that transfusions of whole blood or plasma during hypothermia must be tempered by the possibility of a "rewarming overload phenomenon" especially in the patient with pre-existing cardiac disease.

Whether or not the massive hemolysis in the third patient was due to blood incompatibility is unknown. The possible presence of cold agglutinins in the patient's serum which may have been in part or wholly responsible for the hemolytic phenomenon is speculative. It would seem a reasonable precaution to test for cold agglutinins in all patients in which hypothermia is anticipated.

SUMMARY

1. Experimental evidence suggests that during hypothermia the host immune response remains relatively unaltered whereas bacterial growth and metabolism are retarded. This would seem to justify a clinical trial of hypothermia in the treatment of septic shock.

2. The case histories of three patients in septic shock treated by hypothermia are presented. In all instances hypothermia was utilized only after the failure of patients to respond to accepted therapeutic regimens.

3. A case history illustrating the prophylactic use of hypothermia in infection is presented.

4. Observations of pathophysiologic phenomena occurring during cooling and rewarming are recorded and discussed.

Dr. John M. Allen
Dept. of Surgery
University of Maryland School of Medicine
Baltimore, Maryland

REFERENCES

1. AKIYOMAE, T.: Cited by Whittemore.[28]
2. ALLEN, J. M.: Unpublished data.
3. ALTEMEIER, W. A., AND COLE, W.: Septic shock. Ann. Surg., *143:* 600, 1956.
4. BALCH, H. H., NOYER, H. E., AND HUGHES, C. W.: The influence of hypothermia on experimental peritonitis. Surgery, *38:* 1036, 1955.
5. CURRIE, J.: Cited by Whittemore.[28]
6. D'AMATO, H. E., AND HEGNAUER, A. H.: Blood volume in the hypothermic dog. Am. J. Physiol., *173:* 100, 1953.
7. EBERT, R. V., BORDEN, C. W., HALL, W. H., AND GOLD, D.: A study of hypotension (shock) produced by meningococcus toxin. Circulation Res., *3:* 378, 1955.
8. EISMAN, B., MALETTE, W. G., WOTKYNS, R. S., SUMMERS, W. B., AND TONG, J. L.: Prolonged hypothermia in experimental pneumococcal peritonitis. J. Clin. Invest., *35:* 940, 1956.
9. EZZO, J. A., AND KNIGHT, W. A., JR.: Bacterial shock: a clinical analysis of 38 cases. A. M. A. Arch. Int. Med., *99:* 701, 1957.
10. FAY, T.: Personal communication.
11. FAY, T., AND HENNY, G. C.: Correlation of body segmental temperature and its relation to locating carcinomatous metastasis. Surg., Gynec. & Obst., *66:* 512, 1938.
12. FAY, T., AND SMITH, G. W.: Observations on reflex responses during prolonged periods of human refrigeration. A. M. A. Arch. Neurol. & Psychiat., *45:* 215, 1941.
13. FEDOR, E. J., AND FISHER, B. F.: Effect of hypothermia and rewarming on blood volume determined simultaneously with Cr 51 and T-1824. Fed. Proc., *17:* 45, 1958.
14. FEDOR, E. J., FISHER, B. F., AND FISHER, E. R.: Observations concerning bacterial defense mechanisms during hypothermia. Surgery, *43:* 807, 1958.
15. FEDOR, E. J., FISHER, E. R., LEE, S. H., WEITZEL, W. K., AND FISHER, B. F.: Effect of hypothermia upon induced bacteremia. Proc. Soc. Exper. Biol. & Med., *93:* 510, 1956.
16. FINE, J.: Relation of bacteria to the failure of blood-volume therapy in traumatic shock. New England J. Med., *250:* 889, 1954.
17. FISHER, B., RUSS, C., FEDOR, E. J., WILDE, R., ENGSTROM, P., HAPPEL, J., AND PRENDERGAST, P.: Experimental evaluation of prolonged hypothermia. A. M. A. Arch. Surg., *71:* 431, 1955.
18. FISHER, E. R., FEDOR, E. J., AND FISHER, B.: Pathologic and histochemical observations in hypothermia. A. M. A. Arch. Surg., *75:* 817, 1957.
19. FRIEDMAN, E. W., DAVIDOFF, D., AND FINE, J.: Hypothermia in hemorrhagic shock. Am. J. Physiol., *185:* 521, 1956.
20. HENNEMAN, D. H., BUNKER, J. P., AND BREWSTER, W. R., JR.: Immediate metabolic response to hypothermia in man. J. Appl. Physiol., *12:* 164, 1958.
21. LÖFSTRÖM, B., AND ZEDERFELDT, B.: Wound healing after induced hypothermia. Acta. chir. scandinav., *114:* 245, 1958.
22. MANSBERGER, A. R., JR., OCHSNER, E. W. A., JR., JACOB, S., OPPENHEIMER, J. H., AND GILLETTE, R. W.: A new preparation for the study of experimental shock from massive wounds. II. Evaluation of various therapeutic regimens with special reference to the role of antibiotics, fluid replacement, and debridement. Surgery, *43:* 708, 1958.
23. MORALES, P., CARBERY, W., MORELLO, A., AND MORALES, G.: Alterations in renal function during hypothermia in man. Ann. Surg., *145:* 488, 1957.
24. OCHSNER, E. W. A., JR., JACOB, S. W., AND MANSBERGER, A. R., JR.: A new preparation for the study of experimental shock from massive wounds. I. Standardization. Surgery, *43:* 703, 1958.
25. PETERSON, C. G., AND KRIPPAEHNE, W. H.: Shock in sepsis. Am. J. Surg., *96:* 158, 1958.
26. REEVES, M. M., AND LEWIS, F. J.: Total body cooling in critically ill febrile patients. Surgery, *44:* 84, 1958.
27. TAINTER, M. L.: The effect of low oxygen tensions and temperatures on the actions and toxicity of dinitrophenol. J. Pharmacol. & Exper. Therap., *51:* 45, 1934.
28. WHITTEMORE, W. L., LISA, J. R., AND SAUER, P. K.: Crymotherapy and its relation to hibernation. New York J. Med., *40:* 1563, 1940.
29. WYNN, V.: Electrolyte disturbances associated with failure to metabolize glucose during hypothermia. Lancet, *2:* 575, 1954.

THE MECHANISM OF UTERINE BLEEDING IN THE PRESENCE OF FIBROMYOMAS*

NARINDER SEHGAL, M.B.B.S., AND ARTHUR L. HASKINS, M.D.

Baltimore, Maryland

Uterine fibromyomas, although often asymptomatic, are at times associated with abnormal uterine bleeding. The bleeding, which may be excessive. does not conform to a constant pattern, but varies between hypermenorrhea and polymenorrhea. The absence of a consistent bleeding pattern complicates the differential diagnosis of uterine bleeding in the presence of fibromyomas and necessitates consideration of the other causes of bleeding.

Of theoretic and practical importance in the consideration of abnormal uterine bleeding due to the presence of fibromyomas is the mechanism of bleeding. The most commonly cited cause for "bleeding fibroids" is erosion of the endometrium over the submucous neoplasm with subsequent ulceration and hemorrhage. Other explanations that are offered include compromised contractility of the myometrium, impingement of the myoma upon blood vessels, and venous congestion of the tumor. Gross and microscopic observation of surgical specimens rarely indicate an anatomical cause of the uterine bleeding associated with fibromyomas.

It has been noted that the bleeding difficulties encountered with the uterus containing fibromyomas more frequently occur in the patient in the later years of her reproductive cycle. The patient with ovulatory cycles, fibromyomas and excessive uterine bleeding will ordinarily exhibit hypermenorrhea. The patient with anovulatory cycles, fibromyomas and excessive uterine bleeding will more likely exhibit polymenorrhea. The properly planned administration of estrogenic substance can exert a hemostatic effect on the endometrium in either instance by providing the proper hormonal support.[1] This effect can be maintained over a prolonged period of time if it is essential to the proper preoperative management of the patient.

These facts and observations suggest that the usual mechanism of excessive bleeding in the presence of fibromyomas is not the result of erosion of the endometrium, failure of uterine contractility, or venous congestion. In considering the matter of variations in menstrual flow, it is at once apparent that as the endometrial surface increases in area through the presence of fibromyomas or even as the result of multiparity, the amount of flow increases in the same order. It is also apparent that the amount and frequency of flow may be influenced by hormonal factors and resultant endometrial response.

The purpose of the current study is to correlate the endometrial surface area of the uterus to the presence or absence of uterine fibroids and to the amount of uterine bleeding. Since the patients in the study were selected at random, no control was exercised over the hormonal aspect of any given menstrual cycle. Some of the patients were anovulatory by history and curettage; others were ovulatory by history and curettage.

METHOD

Twenty-six patients were selected from our clinic and hospitalized patients. Hemoglobin determinations were made at the initial clinic visit. Those patients with anemia were placed on iron therapy before hospitalization. Upon being hospitalized each patient was interviewed by one of us for history of abnormal bleeding. Blood studies were repeated. A history of abnormal bleeding or the presence of anemia was the criterion of excessive menstrual flow.

Hysterectomy specimens were obtained immediately upon removal. The endometrial cavity was exposed by a lateral longitudinal incision. Plaster was applied to the endometrial surfaces and was allowed to harden. Upon removal of the plaster, the endometrial surface was identified on the cast with red paint.

When the cast has thoroughly dried, strips of gummed cellulose tape were applied to the endometrial surface of the plaster cast. The sheet of tape was removed and placed on lined

* From the Department of Obstetrics and Gynecology, University of Maryland School of Medicine, Baltimore, Maryland.

TABLE 1

Indications for hysterectomy

Indications	No.	%
Fibromyomas (size only)	8	30
Fibromyomas (excessive bleeding)	13	50
Pelvic relaxation	2	8
Dysfunctional uterine bleeding	2	8
Chronic salpingo-oophoritis	1	4
Total	26	100

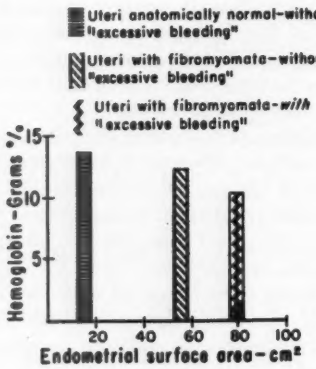

FIG. 1. Endometrial surface area correlated with occurrence of uterine fibromyomas.

paper from which the surface area could be calculated.

RESULTS

Table 1 lists the indications for hysterectomy in these 26 patients. Thirteen of the patients were operated upon because of fibromyomas with excessive uterine bleeding. There was a number of patients who did not give histories of excessive bleeding although they had fibromyomas. Five patients with pelvic relaxation, functional uterine bleeding and chronic salpingo-oophoritis did not have involvement with uterine fibroids and were utilized as examples of a normal endometrial surface. The endometrial surface area in those patients without fibromyomas varied from 11.5 to 18.5 cm.² The average endometrial surface area in the uterus without fibromyomas was 14.5 cm.² The variation in endometrial surface in those patients with fibromyomas was 37.3 to 224.5 cm.²

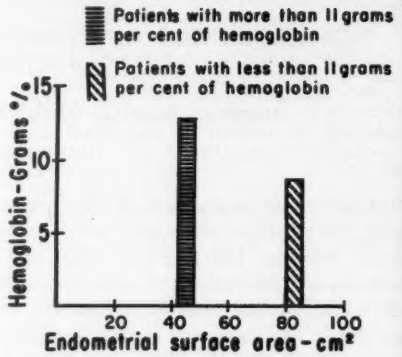

FIG. 2. Initial hemoglobin estimation correlated with endometrial surface area.

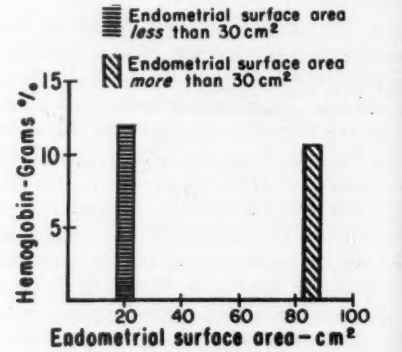

FIG. 3. Endometrial surface area correlated with initial hemoglobin estimation.

The 26 patients were divided into 3 groups: (1) those in whom the uteri was anatomically normal without excessive bleeding; (2) those with uterine fibromyomas without the history of excessive bleeding; (3) those with fibromyomas with a positive history of excessive bleeding. In figure 1 is a graphic representation of the relation between initial hemoglobin levels and endometrial surface in these 3 groups. The patients with anatomically normal uteri and without excessive bleeding, presented with an average initial hemoglobin of 13.5 gm. per cent. The endometrial area was 15 cm.² Those patients with excessive bleeding in the presence of fibromyomas had an average surface area of 80 cm.² and an average initial hemoglobin of 10.5 gm. per cent. The patients with fibromyomas without histories of excessive bleeding occupy an intermediate position in both the surface area,

which is 58 cm.², and hemoglobin, which is 12.1 gm. per cent.

The 26 patients were then divided into 2 groups on the basis of whether the initial hemoglobin was more or less than 11 gm. per cent. Figure 2 represents the correlation between endometrial surface area and initial hemoglobin in these patients. The patients in the group with more than 11 gm. per cent of hemoglobin had an average initial hemoglobin level of 12.5 gm. per cent, and the endometrial surface area was 48 cm.² Those patients with less than 11 gm. per cent of hemoglobin had an average initial hemoglobin level of 8.5 gm. per cent. The surface area of the endometrium in these patients was 87 cm.²

A final division of the patients was again arranged so that two groups were formed; one group in which the endometrial surface area was less than 30 cm.², and the other in which the endometrial surface area was more than 30 cm.² The former group of patients includes the functional uterine bleeders. In figure 3 the findings are represented graphically. The average endometrial surface area in the group with less than 30 cm.² was 22 cm.² The average initial hemoglobin was 12 gm. per cent. These patients with an endometrial surface area of more than 30 cm.² had an average surface area of 87 cm.² and an initial hemoglobin of 10.5 gm. per cent.

CONCLUSIONS

1. The normal endometrial surface area of the uterus in the absence of uterine fibromyomas is about 15 cm.²

2. As the surface area of the endometrium increases as the result of the presence of uterine fibromyomas, the degree of anemia in the patient increases.

3. Although this study does not prove that the mechanism of uterine bleeding in the presence of fibromyomas is due to an increased surface area of the endometrium, it does indicate a correlation between excessive uterine bleeding and increased endometrial surface.

DISCUSSION

Uterine bleeding in the presence of uterine fibromyomas is not necessarily the result of the fibromyomas. It may be the result of other known causes of uterine bleeding including anovulation, cancer, polyps, coagulation defects, or pregnancy. If the bleeding is the result of causes other than fibromyomas, the presence of the fibromyomas do not contraindicate the proper treatment for this specific cause of bleeding. Specific reference in this regard should be made to functional uterine bleeding. Since many of the patients with small uterine fibroids are at or about the time of the climacteric when anovulation and functional bleeding has a high incidence, it should be apparent that the functional uterine bleeding can many times be treated without resorting to hysterectomy simply because of the presence of a small submucous myoma. In addition, if the bleeding in the presence of uterine fibromyomas is excessive and due to the increased endometrial surface area, it may be controlled temporarily by estrogen therapy and allow for the proper preoperative preparation of the patient. This has special significance when considering those patients with relatively severe iron deficiency anemia as the result of chronic uterine bleeding. If the patient is anovulatory, then the administration of graded doses of estrogen will bring the bleeding to a rapid conclusion and allow for the use of iron substances to raise the hemoglobin level prior to surgery. If, on the other hand, the uterine bleeding is ovulatory, the use of graded doses of estrogen to induce anovulation and to promote hemostasis of the uterine endometrium through this mechanism will also allow for the proper alleviation of anemia through the administration of iron.

SUMMARY

The endometrial surface area of 26 patients with various clinical diagnoses was measured. Endometrial surface area in the absence of uterine fibromyomas was less than 20 cm.² In patients with large uterine fibromyomas, the surface area increased to 224.5 cm.² As the surface area increased in this group of patients, the amount of uterine bleeding also increased.

Dr. Arthur L. Haskins
Dept. of Obstetrics and Gynecology
University of Maryland School of Medicine
Baltimore, Maryland

REFERENCE

1. Haskins, A. L.: Adjunctive estrogen therapy. Obst. & Gynec., *11:* 49, 1958.

SPONTANEOUS RUPTURE OF THE ESOPHAGUS

RALPH D. MAHON, JR., M.D., AND LEONARD J. ROBISON, M.D.

Dallas, Texas

It is consistent with the definitions of "spontaneous" and "rupture," as given by Webster[37] and by Dorland,[13] to regard spontaneous rupture of the esophagus as a tearing or breaking apart of the esophagus proceeding from internal impulse or force, without external force. Spontaneous rupture is especially to be distinguished from perforation, a boring or piercing through the esophagus by a benign or malignant disease process, or a traumatic tearing of the esophagus by instrumentation, a foreign body, or other external traumatic force (*e.g.*, compressed air). While the techniques of diagnosis and treatment are similar in rupture and perforation of the esophagus, the two conditions differ markedly in several respects. Perforation has no consistency as to site or axial orientation of the esophageal wound, whereas rupture almost invariably occurs as a longitudinal rent in the left lower esophagus just above the diaphragm. Where there has been instrumental or other obvious trauma to the esophagus, the correct diagnosis of perforation is usually made; whereas, in cases of spontaneous rupture, one is less likely to consider the correct diagnosis. Furthermore, since most cases of spontaneous rupture are associated with vomiting, there is greater contamination than in perforation, and a more rapid, severe collapse ensues. Although some authors have used, with excellent rationale, such terms as "postemetic" and "juxtadiaphragmatic" rupture of the esophagus, most authors have used the designation, "spontaneous rupture of the esophagus," and have defined it essentially as we have in selecting cases for reporting or reviewing.

INCIDENCE

The initial description of rupture of the esophagus was by Boerhaave in 1724. The condition has been infrequently reported. Derbes and Mitchell[12] in 1956 reviewed 157 reported cases and added 14 cases; all but one of their own cases were diagnosed at autopsy. Barrow and Thompson[5] in 1957 wrote that about 160 cases had been described in the world literature to date. Anderson[4] in 1957 stated that nearly 200 cases of this condition had been collected without an attempt to exhaust the literature thoroughly.

We believe that it occurs more commonly than the number of reported cases would suggest. It is likely that a significant number of cases have been diagnosed but not reported, or have died, incorrectly diagnosed, and have escaped autopsy.

The following considerations support this belief. Many of the reported cases were diagnosed correctly only at autopsy. Carter,[10] at the University of Virginia, found an incidence of 1 ruptured esophagus to every 72 perforated duodenal ulcers admitted between 1940 and 1949. When Starkey,[32] at a meeting of the American Association of Thoracic Surgery, reported 4 cases seen within 18 months, 7 others present mentioned 25 cases treated personally. Swann[33] reported 4 cases in one medical community within 2 years. Samson[30] reported 2 cases and knew of 2 others, all occurring in the Oakland area within 6 months. We have heard of 2 unreported recent cases in Dallas.

DIAGNOSIS

Spontaneous rupture of the esophagus is not ordinarily a difficult diagnosis to establish if the physician is but aware of the condition and thinks of it. It most frequently occurs in a middle-aged or older person, 5 times as likely to be male as female. The patient may have preexisting or coexisting disease of some part of the gastrointestinal tract. The rupture is usually announced by sudden, severe pain following emesis, which may contain blood, and may follow heavy food or alcohol intake. The pain is usually located in the epigastrium and precordium, with radiation around the lower chest or to the back. Often a sensation of something tearing, popping, bursting, or giving way is described. The pain, often so severe as to resist narcotic relief, is aggravated by breathing, talking, swallowing, or moving about, and the patient abstains from a reclining position.

Usually dyspnea and shock of variable degree ensue rather rapidly.

One usually finds the patient in severe, acute distress, in a sitting or semisitting position. There are usually abdominal rigidity and tenderness, which may be limited to the upper abdomen. The voice may have an altered quality or nasal twang. Cyanosis, hypotension, dyspnea, and tachycardia are usually present. Subcutaneous emphysema may be detected in the neck. Chest examination may reveal mediastinal widening, a mediastinal crunch or click synchronous with the heartbeat, or signs of pleural effusion, pneumothorax or hydropneumothorax.

X-ray examination is the most helpful diagnostic technique. Posteroanterior and lateral views of the chest should be requested. These may reveal mediastinal emphysema, mediastinal effusion, retrocardiac gas, gas outlining the juncture of the mediastinal and diaphragmatic pleurae (V sign of Naclerio[25]), pleural effusion, pneumothorax or hydropneumothorax. The chest abnormalities may be bilateral. Subdiaphragmatic gas has never been described in rupture of the esophagus. A swallow of radiopaque liquid may confirm the diagnosis of esophageal rupture and reveal the site of the leak.

If pleural effusion is present, thoracentesis may yield fluid suggestive of emesis and having an acid reaction. If oral dye has been administered, it may be recovered by thoracentesis.

Diagnoses which have been made erroneously in the presence of esophageal rupture include perforated peptic ulcer, coronary occlusion, pancreatitis, mesenteric thrombosis, dissecting aortic aneurysm, high intestinal obstruction or strangulation, diaphragmatic hernia, spontaneous pneumothorax, bronchopleural fistula and pulmonary embolism.

Mackler[21] said that a triad of vomiting, low thoracic pain and emphysema of the neck is diagnostic. Barrett emphasized a triad of rapid respiration, abdominal rigidity, and subcutaneous emphysema. Samson[30] felt that the history and findings in many cases are typical enough for the diagnosis to be made by telephone. Bunch urges that "every case of suspected perforated peptic ulcer should have erect x-ray of the chest. . . . If there is esophageal rupture, some abnormality will be seen on the chest films in 90 per cent of the cases, and no free air will be seen beneath the diaphragm."

PATHOLOGY

Almost invariably, the site of rupture has been in the left lower esophagus just above the diaphragm. Except for the transverse ruptures reported by Boerhaave and by Higginson and Clagett, it has been a longitudinal rent. The susceptibility of the site has been attributed to the buttressing of the right side of the esophagus by the vertebral bodies and the pericardium, the protection afforded the short subdiaphragmatic esophagus by the liver and the gastric fundus, the absence of voluntary muscle in the lower third of the esophagus, the curve of the lower esophagus, and the points of entrance of esophageal vessels. Experimental rupture by air or hydrostatic pressure of human esophagi *in situ*[6, 12] and of isolated human esophagi[21] has demonstrated the marked tendency for the esophagus to rupture at this site. Mackler's[21] work suggests intrinsic weakness at this point. It is apparent from the pressures at which the esophagi ruptured that vomiting may produce sufficient pressure to rupture the esophagus, especially if egress of a large volume of food or fluid from the oral end of the tube is interfered with by food bolus, stricture, cricopharyngeal spasm or alcoholic incoordination.

The longitudinal orientation of the tear is explained by Derbes and Mitchell's[12] complicated mathematical analysis, using the principles of free body diagrams, which shows that "mechanical (hydrodynamic) factors incident to increased internal pressures compel the esophagus to tear in a vertical plane."

Microscopic examination of the esophagus at the site of the rupture may show no abnormality, or may reveal variable degrees of ulceration, necrosis and cellular infiltration.

Usually the spilled gastric contents are contained under pressure in the mediastinum for a variable time, the gas dissecting upward toward the neck, followed by perforation of the mediastinal pleura and extension to the pleural cavity.

PROGNOSIS AND TREATMENT

Fojcik[14] refers to the case of Sandberg, reported in *Acta chirurgica scandinavica*, 1955, promptly diagnosed, and treated by nasogastric suction, thoracenteses and intrapleural antibiotics with recovery. Other than this case, no survival has been reported without operative intervention. Derbes and Mitchell[12] state that

without operation, 25 per cent die within 12 hr. of rupture, 65 per cent die within 24 hr., 89 per cent die within 48 hr., and none lives longer than 1 week.

The first cure was achieved in 1911 by Sencert, who repaired, via laparotomy, a rupture of the abdominal esophagus. Transabdominal repair of the ruptured esophagus has been reported two times since; Foggitt's patient died, and Hunt's[18] survived. The first cures in this country were Overholt's case, who, in 1943, had left posterior mediastinotomy and bilateral pleural drainage, and two cases, mentioned in an editorial comment by Graham in the 1944 *Yearbook of Surgery*, who survived with empyema drainage.

The first thoracotomy and repair of ruptured esophagus was the unsuccessful operation reported by Collis in 1944. The first successful cases of thoracotomy and repair were reported in 1947 by Barrett and by Olsen and Clagett.[26] The patient of Olsen and Clagett was promptly diagnosed, operated upon 3 hr. postrupture, and had a benign, rapid convalescence.

In their 1956 review of 157 cases of ruptured esophagus, Derbes and Mitchell[12] found that through 1952, operation had been performed in 55 cases, with 35 survivals. Of these, we have counted 35 cases who had repair of the rupture, with 25 survivals, a mortality rate of 29 per cent. In the 20 cases who had drainage procedures, without esophageal repair, there were 10 survivals, a mortality rate of 50 per cent.

From the post-1952 articles listed in the references, admittedly not an exhaustive review, we have collected reports of 42 additional cases of ruptured esophagus; none survived without operation. In the 13 cases who had drainage procedures, without esophageal repair, there were no survivals. In the 16 cases having repair, there were 11 survivals, a mortality rate of 31 per cent.

It is interesting to compare earlier statements regarding treatment with more recent ones. Frink,[15] whose patient recovered with closed and open chest drainage, wrote in 1947 that "surgical repair of the esophagus . . . is truly a formidable procedure to perform on a desperately ill patient, and, to be successful, would have to be done within a few hours after the rupture had occurred, when an accurate diagnosis is most difficult." He suggested "surgery limited to simple drainage of the infected region." Moore and Murphy,[23] whose patient recovered with repair of the esophageal laceration by esophagogastrostomy 61 days postrupture, said in 1948, "We question the wisdom of immediate repair of the ruptured esophagus in those patients whose desperate condition following the accident is a strong contraindication to any major surgical procedure." These statements seem rather naive today.

Samson,[30] Alt,[2] and others have emphasized the importance of early operation as a resuscitative measure, calling attention to the remarkable improvement in the patient's condition as soon as the bulging mediastinal pleura is incised and the mediastinum decompressed. The presence of shock in esophageal rupture should not delay operation. Drainage procedures are considered to have no proper place in the early period; they are of value only in those patients who fortuitously survive the first few days and have localized infection.

Optimal treatment of esophageal rupture consists of prompt diagnosis and institution of resuscitative measures; early left thoracotomy, usually through the bed of the 7th or 8th rib; wide opening of the mediastinal pleura; closure of the esophageal tear in two layers with interrupted nonabsorbable sutures; meticulous cleansing of the mediastinum and pleural cavity; and intercostal catheter chest drainage, with the mediastinal pleura left open. In patients operated upon at a later stage, decortication may be necessary for adequate lung re-expansion. Nasogastric suction, adequate antibiotics, oxygen, and indicated intravenous fluid therapy are employed.

Parenthetically, the efficacy of similar treatment in traumatic perforation of the esophagus is reported by Weisel and Raine.[38] Their 7 patients were diagnosed at intervals of 3 to 38 hr. after injury, were treated by closure of the perforation and drainage of the fascial or pleural space, antibiotics and supportive therapy, with 100 per cent survival.

CASE REPORT

A 60-year-old white man was seen at the East Dallas Hospital, a 35-bed general hospital, at 3:00 p.m. on February 23, 1958. At about 1:30 p.m., after eating Sunday dinner, he became nauseated and vomited twice, the 2nd emesis containing a little blood. He "felt something pop" and had the onset of severe, cramping upper abdominal pain with interscapular radiation. The pain became so severe that it inhibited breathing and prevented

reclining. He had noted some epigastric burning pain for about 2 months.

The patient was in a semisitting position and obviously in severe distress. Examination was restricted by his extreme pain, aggravated by manipulation or attempting to recline. The skin was pale and moist; temperature was 97°; blood pressure was 190/120; pulse rate was 96 per min.; and respirations were 22 per min. The abdomen appeared generally rigid, especially in the epigastrium, where there was marked tenderness. Peristaltic sounds were absent. Cursory chest examination was not remarkable.

Urinalysis and blood count were not remarkable. Suboptimal x-ray of the lower chest and upper abdomen showed no subphrenic gas, but there appeared to be free gas about the lower pole of the spleen. That part of the chest visible on the film appeared normal.

The patient was thought to have a perforated peptic ulcer, and no other diagnoses were seriously entertained.

Operation was begun at 5:45 p.m., 4 hr. after onset, through a right upper quadrant, obliquely transverse, laparotomy incision. Careful abdominal exploration was negative except for an esophageal hiatus enlarged to 5 cm. and having a boggy feel. Considering that the abdominal catastrophe might have been transient incarceration of the stomach in the hiatus hernia, the abdominal incision was extended to the left costal margin for hiatus hernia repair.

Upon opening the peritoneum at the hiatus, one entered a large mediastinal collection of content of a recent meal, including meat and salad. A rent in the lower esophagus was palpated. The chest was rapidly opened by extending the incision through the 7th intercostal space as far as the anterior axillary line. The diaphragm was incised through the esophageal hiatus. The left pleural cavity contained several hundred cubic centimeters of serous fluid. After dividing the inferior pulmonary ligament, the intact mediastinal pleura was extensively incised.

There was a 3-cm. longitudinal rupture of the left anterolateral aspect of the esophagus, just above the level of the diaphragm. The esophageal wall appeared entirely normal. The gastric contents had dissected a cavity in the left posterior mediastinum to the level of the aortic arch. There was a grayish discoloration of the mediastinal tissues.

The esophagus was repaired with 2 layers of interrupted fine cotton, the inner row with knots to the lumen, and the outer row of Halsted mattress sutures. The mediastinum and pleural cavity were meticulously cleansed of food particles and throughly irrigated with warm saline. The mediastinum was drained through a paravertebral intercostal stab wound, the pleural cavity by intercostal water-seal catheter, and the left subphrenic space through a left lateral abdominal stab wound.

The diaphragm and hiatus were repaired with interrupted cotton except for a chromic catgut suture immediately adjacent to the esophagus. The thoracoabdominal incision was closed in layers with interrupted cotton, plus pericostal sutures of chromic catgut. Rather poor approximation of the costal cartilage was obtained with a figure-of-eight suture of no. 28 stainless steel wire. Whole blood, 500 cc., had been given during the procedure.

The postoperative orders included digitalization, antibiotics, vitamins, intravenous fluids, nasogastric suction, and intermittent positive pressure inhalations. Ambulation was begun 10 hr. postoperatively. Nasogastric suction was discontinued on the 4th postoperative day. Oral intake was begun on the 5th postoperatve day after Gastrografin swallow demonstrated no esophageal leak. The abdominal drains were removed on the 4th, the mediastinal drains on the 6th, and the intercostal catheter on the 8th postoperative day. Skin sutures were removed on the 8th postoperative day.

On the 9th postoperative day, dehiscence of the abdominal portion, costal margin and superficial thoracic portion of the thoracoabdominal incision occurred. There was no apparent communication with the free pleural cavity. The wound disruption was closed with through-and-through sutures of no. 28 stainless steel wire.

On the 15th postoperative day, 100 cc. of serum were drained from the posterior end of the incision; a small amount of serous, occasionally seropurulent, drainage occurred intermittently for the next 2 weeks. Whole blood, 500 cc., was given on the 16th and on the 17th postoperative days. Temperature rose to 100 to 101° on the 1st through the 20th postoperative days. The patient was discharged from the hospital on the 27th postoperative day, and the wire sutures were removed 3 days later.

The patient has done well since. There is no incisional hernia. There is instability of the left costal margin without paradoxic motion. Upper gastrointestinal x-ray examination on November 24, 1958, was normal; the esophagus showed no narrowing. Chest fluoroscopy on December 22, 1958, was not remarkable except for an elevated, paralyzed left diaphragm with paradoxic motion.

COMMENT

We are chagrined that the diagnosis of esophageal rupture was not considered preoperatively.

The patient's history was typical. Adequate chest x-rays and radiopaque swallow almost certainly would have established the diagnosis. Thoracotomy and repair would have been a more rapid and efficient operative procedure; morbidity would have been less; wound disruption would have been unlikely; and hospital stay would have been shorter. Of course, all concerned are grateful for the patient's recovery.

SUMMARY

Since Boerhaave first described the condition in 1724, some 200 cases of spontaneous rupture of the esophagus have been reported, with some 50 survivals. All those cured, save one, had operative intervention of some sort. The lowest mortality rate was in those patients having thoracotomy with repair of the esophagus.

Failure to make the diagnosis of esophageal rupture is due to failure to consider it. One should always think of it in patients developing severe epigastric or chest pain after vomiting; in patients presenting both abdominal and chest findings; in patients thought to have perforated peptic ulcer; in patients with cervical emphysema; in patients who present a shocklike state following vomiting; and in patients with hydropneumothorax. The diagnosis is confirmed by the demonstration of esophageal leak after swallowing radiopaque material.

Early diagnosis, followed by thoracotomy, repair of the esophagus, and pleural drainage, may be expected to give a favorable outlook in a condition almost uniformly fatal otherwise.

A case of spontaneous rupture of the apparently normal esophagus is reported. Preoperative diagnosis was incorrect, but early primary repair of the esophagus was effected, and recovery was accomplished, despite dehiscence of the thoracoabdominal incision.

Whenever abdominal exploration for suspected perforated peptic ulcer is negative, the possibility of esophageal rupture should be considered. Immediate steps should be taken to ascertain its presence, most importantly, chest x-ray with swallow or instillation of radiopaque fluid.

Dr. Ralph D. Mahon, Jr.
6003 Victor
Dallas, Texas

REFERENCES

1. ABEL, W. G.: Spontaneous rupture of the esophagus. Am. J. Surg., *94:* 663, 1957.
2. ALT, R. E., BIXBY, F. E., AND ROWELL, R. J.: Spontaneous rupture of the normal esophagus. New England J. Med., *249:* 1060, 1953.
3. ANDERSON, R. L.: Rupture of the esophagus. J. Thoracic Surg., *24:* 369, 1952.
4. ANDERSON, R. L.: Spontaneous rupture of the esophagus. Am. J. Surg., *93:* 282, 1957.
5. BARROW, G. J., AND THOMPSON, W. T., JR.: Spontaneous rupture of the esophagus. South. M. J., *50:* 95, 1957.
6. BODI, T., FANGER, H., AND FORSYTHE, T.: Spontaneous rupture of esophagus. Ann. Int. Med., *41:* 553, 1954.
7. BROOKER, R. M.: Spontaneous perforation of the esophagus with report of a case. Am. Surgeon, *22:* 129, 1956.
8. BUNCH, G. H., JR.: Spontaneous rupture of the esophagus. Ann. Surg., *145:* 1001, 1957.
9. BUSHEY, H. L.: Rupture of the esophagus; report of two cases with atypical features. New England J. Med., *255:* 1146, 1956.
10. CARTER, J. P., ALRICH, E. M., AND DRASH, E. C.: Spontaneous rupture of the esophagus. Surgery, *30:* 500, 1951.
11. CHRISTOFORIDIS, A., AND NELSON, S. W.: Spontaneous rupture of esophagus; with emphasis on the roentgenologic diagnosis. Am. J. Roentgenol., *78:* 574, 1957.
12. DERBES, V. J., AND MITCHELL, R. E., JR.: Rupture of the esophagus. Surgery, *39:* 688, 865, 1956.
13. DORLAND, W. A. N.: *The American Illustrated Medical Dictionary*, Ed. 18. W. B. Saunders Company, Philadelphia, 1938.
14. FOJCIK, K. J.: Juxtadiaphragmatic rupture of the esophagus. Illinois M. J., *112:* 227, 1957.
15. FRINK, N. W.: Spontaneous rupture of the esophagus. J. Thoracic Surg., *16:* 291, 1947.
16. HAYES, D. W.: Spontaneous rupture of the esophagus. South. M. J., *46:* 962, 1953.
17. HEWLETT, T. H., AND BUCHANAN, A. C.: Esophageal rupture associated with peptic ulcer. Am. Pract. & Digest Treat., *8:* 1733, 1957.
18. HUNT, E. L.: Spontaneous rupture of the esophagus with successful repair by subdiaphragmatic approach. Am. Surgeon, *21:* 1158, 1955.
19. KRAEFT, N. H., AND HUGHES, F. A., JR.: Rupture of the normal esophagus. Surgery, *31:* 291, 1952.
20. LAYNE, J. A., AND HILDEBRAND, E.: Rupture of the esophagus following severe vomiting. Gastroenterology, *13:* 170, 1949.
21. MACKLER, S. A.: Spontaneous rupture of the esophagus. Surg., Gynec. & Obst., *95:* 345, 1952.
22. MIGLIACCIO, A. V., FORSYTHE, T., AND CAVANAUGH, C.: Spontaneous rupture of the esophagus. Surgery, *36:* 826, 1954.
23. MOORE, J. A., AND MURPHY, J. D.: Spontaneous rupture of the esophagus; report of 1 case, with recovery. J. Thoracic Surg., *17:* 632, 1948.
24. MUENDEL, H. J., AND LEVISON, W.: Spontaneous rupture of the esophagus. A. M. A. Arch. Surg., *67:* 943, 1953.
25. NACLERIO, E. A.: The "V sign" in the diagnosis of spontaneous rupture of the esophagus (an early roentgen clue). Am. J. Surg., *93:* 291, 1957.

26. Olsen, A. M., and Clagett, O. T.: Spontaneous rupture of the esophagus; report of a case with immediate diagnosis and successful surgical repair. Postgrad. Med., *2:* 417, 1947.
27. Pate, J. W., Hughes, F. A., and Patton, T. B.: Spontaneous rupture of the esophagus. Am. Surgeon, *24:* 385, 1958.
28. Paton, B. C.: Spontaneous rupture of the esophagus. Scottish M. J., *2:* 97, 1957.
29. Priviteri, C. A., and Gay, B. B., Jr.: Spontaneous rupture of the esophagus; with report of 5 cases. Radiology, *57:* 48, 1951.
30. Samson, P. C.: Postemetic rupture of the esophagus. Surg., Gynec. & Obst., *93:* 221, 1951.
31. Small, A. R., and Boyd, L. J.: Rupture of the esophagus induced by vomiting. Am. J. Digest. Dis., *19:* 73, 1952.
32. Starkey, G. W. B.: Spontaneous rupture of the esophagus with presentation of 4 new cases. J. Thoracic Surg., *30:* 300, 1955.
33. Swann, W. K., Bradsher, J. T., Jr., Lomasney, T. L., and Retterbush, W. C.: Spontaneous rupture of the esophagus; a not uncommon medical emergency. J. Tennessee M. A., *48:* 407, 1955.
34. Wachtel, F. W., and Genkins, G.: Spontaneous rupture of the normal esophagus; report of a case with an unusual clinical course and pathologic findings. J. Mt. Sinai Hosp., *22:* 6, 1955.
35. Ware, G. W., Shnider, B. I., and Davis, E. W.: Spontaneous rupture of esophagus; report of a successful case with cardiac arrest and review of the literature. A. M. A. Arch. Surg., *65:* 723, 1952.
36. Webster, P. D., III, and Taylor, J. R.: Traumatic rupture of the esophagus by compressed air; report of case with review of 110 cases of esophageal rupture. North Carolina M. J., *18:* 305, 1957.
37. Webster, N.: *Collegiate Dictionary*, Ed. 5. G. & C. Merriam Company, Publishers, Springfield, Mass., 1940.
38. Weisel, W., and Raine, F.: Surgical treatment of traumatic esophageal perforation. Surg., Gynec. & Obst., *94:* 337, 1952.

THE USE OF HYPNOANALGESIA IN THE RELIEF OF INTRACTABLE PAIN

MAURICE M. TINTEROW, M.D., F.A.C.A., F.I.C.S.

Wichita, Kansas

Pain is a sensation which is dependent on the consciousness.[1] It is a well known fact that distraction from attention may exclude from its consciousness many sensations including that of pain. Pain which is perceived is subjective and individual, and it is an emotional as well as a sensory experience.[18] It varies in accordance with the emotional and physical makeup of the patient. Variations in the perception of pain, its minimization, distortion or exaggeration, occur because of associated attitudes and emotions.[15] Words have long been recognized as exerting a tremendous influence on human emotions, and hypnosis as a state of hypersuggestibility provides an excellent means of influencing sensory and perceptive functions. Since pain may originate through the activity of the emotional and intellectual functions of the thalamus and cerebral cortex, hypnosis has been used as a means of achieving relief of this condition. In some cases following complete investigation, although the origin of the pain can be properly localized, the cause cannot be determined, nor can the failure to obtain relief be explained. In many patients the pain is not traceable to any causative factor, and personality difficulties with states of tension, anxiety, and other emotional disturbances may become so prominent as to completely mask the primary cause of pain. Because the emotional factor may be so prominent in the patient with intractable pain, hypnosis can be used rather than the usual surgical and medical methods of relief. These methods can be used in the event of failure of the hypnotherapy.

Reference to the use of hypnosis in the relief of pain and emotional states has occurred throughout the history of medicine. However, it was during the 19th century that hypnosis was first understood. The "animal magnetism" of Franz Anton Mesmer is known to all, and through it hypnosis fell into disrepute. James Esdaile in 1850 published a report of 300 major and thousands of minor procedures performed in India under hypnosis with amazing results. He was not recognized by the Royal Society of Medicine in England, and with the advent of chemical anesthesia, hypnosis again declined in popularity. James Braid, an English surgeon, was the first to report that hypnosis was a utilization of suggestion, but did not realize that it was the explanation of the underlying process. Charcot, Liebault, and Bernheim had schools of hypnotherapy in France, but Freud's rejection of hypnosis in favor of his own theories relegated hypnosis back to the stage of amusement and mystery. During the last 10 years interest has again been stimulated in England and in this country with research and clinical studies. Today it is accepted as an ethical part of the practice of medicine.

The impressive bewildering character of hypnotic manifestations and the profoundly inexplicable seemingly miraculous, psychologic effects upon human behavior achieved by the use of hypnosis, have served to bring about contradictory attitudes toward it. The unscientific attitude of superstitious awe, fear, disbelief and actual hostility have delayed and obstructed the growth of scientific knowledge of hypnosis.[8] If physicians today understood more clearly what hypnosis is and what could be expected of it, then it would be more widely used. Hypnosis is a technique which in skilled hands has very little danger and its application, indications and contraindications can become a part of the knowledge of the practicing physician.[2]

Success in hypnosis depends on establishing selective thinking in a patient with the bypass of the critical factor. It raises the threshold of pain and is the only means of anesthesia which carries no danger to the patient. It is a pleasant experience involving no tension or apprehension, and it can be maintained for a long period of time. Hypnosis places no extra load on the circulatory, respiratory, hepatic, or renal systems. Hypnosis obeys the laws which govern conditioning in general, and suggestion causes new conditioned patterns to be developed. A suggestion is an idea accepted by an individual without resistance or criticism and unanalytically, and upon which he therefore acts.[9] There are as many ways of producing hypnosis as there are

hypnotists, and it is not the aim of this paper to try to teach techniques. Techniques and methods of hypnotic induction can be found in many of the textbooks dealing with the subject, and many of the techniques are combined by the individual using hypnosis to fit the individual patient.[10, 14, 17, 19] Success in hypnotherapy depends on the ability of the individual and, of more importance, on constant practice. It is of utmost importance that the patient believe the competency and integrity of the hypnotizing physician. Under hypnosis there seems to be a dependency of the patient on the physician, and in the hypnotic state the subject tends to carry out the suggestions in order to please the physician. Under the influence of hypnosis the sensory end organs continue to function but the patient's attitude may be so altered that the pain is no longer experienced in the same way.

Patients with intractable pain need mental and physical therapy more than any other group of patients. Intractable pain is not responsive to conservative measures. It is usually produced by a disease which cannot be treated directly.[12] Drug therapy will usually give relief, particularly opium and its derivatives, but with continued usage the severity of the pain has no relationship to the amount of narcotic necessary. The reaction of the patient to pain is dependent upon the attitude and mood of the patient. Suggestive therapy has been tried with the use of placebos, but it has been shown that only those of high suggestibility receive benefit from their use. Hypnosis is nothing more than the suggestive placebo effect presented in a specific interpersonal setting.[5]

The application of medical and surgical methods for the relief of pain had its inception early in medical history. Many surgical procedures have been devised for relief of intractable pain; however, these have not been accepted as desirable because of the destructive nature. Such procedures as prefrontal lobotomy or thalamotomy in nonpsychotic individuals will relieve the physical suffering but will also produce a marked blunting of the emotions and of the initiative as well as a lowering of the intelligence. Hypnosis has been suggested to be a "psychologic" lobotomy, but hypnosis was tried before and after prefrontal lobotomy, and it did not make the patient more easily hypnotized as it should if it were a surgical and permanent form of the same state of mind.[3] Others, such as neurectomy, rhizotomy, chordotomy, and ablation of the sensory cortex, have been developed as a means of interrupting the sensory pathways to the conscious centers. However, if drug addiction has been present, these operations do not have the results they would have if they had been performed earlier. The mental reaction to pain causes the deterioration of the patient as much as the pain itself does. Under a majority of circumstances, pain serves as a protective physiologic sensation evoking bodily reactions which prepare the individual for either fight or flight as a result of stimuli arising from the surface, or conversely, immobilization, as a result of stimuli arising in the viscera. However, severe unremitting pain may be a pathologic process destructive to the organism.[11]

Nearly all of the patients with intractable pain reach debilitating states through exhaustion, loss of sleep, decrease of appetite and weight loss. The use of hypnotherapy for correction of the underlying emotional basis in these patients is advisable, because it contributes to the perpetuation of the illness. Much of the pain may occur as an excessive reaction to a slight stimulus, or as a guilt complex to a previous situation. The use of suggestive therapy should be attempted early and before addiction to narcotics or alcohol has occurred. It is undesirable to resort to the use of narcotics in stupefying doses for the relief of such pain when the duration of life may be several months.[4] Relief of the severe pain may be secured at the first session of suggestive therapy, and an attempt is made to secure as deep a relaxation as is possible. The word hypnosis is not used because of the fear the patient has toward the mystery and the unknown. The term "medical relaxation" is referred to as the method of therapy being used for the relief of intractable pain.

There are many methods of inducing the trance state in a patient. The oldest is the fixation method whereby the patient looks at a fixed point or a light until the eye muscles tire and through suggestions by the operator cause the eyes to remain closed. Many of the methods which are described and used are time consuming. However, since all patients respond in a different manner, varying techniques are utilized by this author. Progressive relaxation is suggested with eye closure and deep sleep being the end results. Amnesia is established, and the patients reach different depths of relaxation

dependent upon their ability to achieve a trance state. In order to achieve a trance state the patient must have the ability to concentrate, the desire to be hypnotized, the ability to take suggestion, and absolute freedom from fear. The presence of fear in the patient is responsible for the inability of the patient to become suggestible. There are certain signs which may be elicited during hypnosis, and if they are present, the patient has definitely entered into the state of hypnosis. These signs may be fluttering of the eyelids, injected conjunctiva, tearing of the eyes, physical relaxation, and catalepsy of the limbs. Continued training and education adequately prepares the patient. There are three states which may be reached during hypnotic sessions: (1) The light or superficial trance in which the patient accepts the suggestion for relief of pain while in the trance, but it is of no value after being brought out of the trance. (2) The deep trance in which posthypnotic suggestions for the relief of pain are successful after the patient is awakened from the trance state. (3) Plenary or stuporous trance in which the patient reaches such a state of relaxation that there is marked slowing of all organic responses and is of no value because suggestions will not be accepted by the patient.

The depth of hypnosis varies with the experience and ability of the operator. Hypnotic techniques will reduce pain, allay anxiety, and aid in organ function, but the results are proportional to the depth of hypnosis achieved. Only those patients who can reach a deep trance can benefit from this therapy. It is in this state that the emotional tension present in these patients can be relieved, and neurotic exaggeration and self-pity can be exchanged for a more constructive and realistic point of view. There is a decrease in the degree of dread which surrounds a patient with intractable pain.

Hypnoanalysis is attempted with all patients receiving therapy. The possibility of a traumatic experience entering into the emotional conflict of the patient is always present and should be elicited if at all possible. When abreaction of some relevant event in the patient's life was indicated, this was discussed while the patient was in the deep trance, then brought up to a conscious level and discussed again. The removal of a traumatic experience or guilt complex greatly enhanced the possibility of success of the posthypnotic suggestion.

The advantages of hypnotherapy far outweigh the disadvantages. Drug requirements are lessened and may be removed completely from the patient. The organ dysfunction is corrected, appetite is increased, and the desire to live becomes apparent. Depression, anxiety, and the fear of impending death, particularly in those patients with inoperable and metastatic carcinoma, is removed, and the patient approaches a normal frame of mind. He is a rational thinking human, rather than a vegetating invalid as may happen after a prefrontal lobotomy. The patient is supported in his hour of need by the compassion of his physician rather than let down with drug addiction and destructive surgery.[3]

The disadvantages should not deter a competent and understanding physician from attempting to secure relief of intractable pain. Many of the patients, because of the severity of the condition, are not good subjects and do not reach the proper depth for posthypnotic suggestions to be effective. Hypnotherapy is time consuming on the practicing physician, and the number of trained physicians are still few in number. Many physicians still have an aversion to its use and resort to the use of large amounts of narcotics to make the patient comfortable. Public resentment to the use of hypnosis still persists, and proper education has to be undertaken before it is an accepted medical procedure. Psychotherapy of any type can, of course, be exceedingly dangerous to the patient if its practitioner is incompetent.[16]

Hypnotherapy was attempted in 15 cases of intractable pain. Success depended upon the early referral of the cases and before addiction to narcotics had occurred. Five of the eight cases with metastatic carcinoma were relieved of the severe intractable pain. Of two patients with intractable pain following herpes zoster, relief was successful in one of them. A patient with diabetic neuropathy had no pain after five sessions of hypnosis. After an operation for repair of a hiatal hernia, the patient had severe intractable pain which was relieved with hypnotic therapy. Another patient, who developed pain after a subtotal gastrectomy, was also successful in returning to a normal life. Two other patients, one with a complaint of pain after an automobile accident, and another with a phantom limb were unable to be hypnotized and considered failures. The following case reports demonstrate

the types of cases in which hypnotherapy was considered and attempted.

CASE REPORTS

Case 1. A 32-year-old white woman was found to have a carcinoma of the cervix in 1954. Radiation therapy followed by a hysterectomy was the treatment of choice. There was an onset of pain in the left thigh and left hip in March 1957. In September 1957, a mass was discovered in the left pelvis. X-rays revealed a metastatic lesion to the left pelvis. Cobalt therapy was instituted. By March 1958, there was a loss of 22 lb. The patient had almost continuous vomiting and was receiving 50 mg. of Sparine and 100 mg. of Demerol every 2 hr. because of severe pain and a depressed state. Suggestive therapy was started on April 17, 1958. The same night the vomiting ceased, and the patient was mentally improved. No opiates were given that night, and the patient's general condition improved. Further hypnotherapy was given for 7 days, and the patient received no further narcotics during this time. The differentiation of anticipation and pain as separate entities, and the use of time distortion was of value in the therapy of this case.[7] At the end of the 4th day, the patient's appetite was improved, and she was sitting up in a chair. Only an occasional Empirin compound was needed for a headache. She was dismissed from the hospital with complete freedom of pain in the left hip. She was last admitted to the hospital on November 29, 1958, still receiving no opiates although x-rays revealed further increase in size of the metastatic lesions. There was also lymphatic obstruction due to the metastatic carcinoma. She has gained 16 lb. and there is still a favorable mental outlook. She was discharged from the hospital after receiving further reinforcement of the hypnotic suggestions for the relief of pain.

Case 2. A 45-year-old white man entered the hospital on November 23, 1951, and was found to have a lymphosarcoma of the spleen and cervical nodes. The spleen was removed, and later a radical neck dissection was performed. He was given nitrogen mustard for the generalized lymphosarcoma. On April 7, 1958, he had sudden severe pain in the left temporal and occipital areas. A diagnosis of herpes zoster, left C-3 was made. His pain was constant after disappearance of the lesions with limitation of motion of the head. Suggestive therapy was instituted on June 26, 1958, with deep somnambulism being achieved on the second session. Time distortion was utilized with some success. Relief was obtained with posthypnotic suggestion, and this was reinforced weekly up until the time of his death on August 21, 1958.

Case 3. A 49-year-old white woman developed diabetes mellitus in 1949. She had several admissions for diabetic acidosis. Her last admission was August 19, 1958, for acute pain of the left lower extremity primarily centered about the knee. The pain was not aggravated by walking, but remained quite severe, being unrelieved with 100 mg. of Demerol. A diagnosis was made of diabetic neuropathy with possible complication of arthritis. She continued to have intermittent episodes of severe pain and required narcotics about every 2 hr. for relief. Hypnotherapy was started on November 3, 1958, and daily sessions were attempted for 6 days. Relief from intractable pain was noticed after the 3rd session and no further opiates were needed. Since the suggestive therapy was completed, reinforcement was necessary after 2 weeks, and she has had no further incapacitation due to pain since that time.

The above cases cite examples of illnesses in patients in whom hypnotherapy was indicated and successful. Time does not permit discussion of all 15 cases, and those in whom failure was present had underlying emotional states and dependencies which could not be corrected even with abreaction and hypnoanalysis. Pain, like other psychologically based symptoms, is frequently utilized as justification and rationalization for drug habituation or addiction, or for the operative intervention for which some patients have so great a need in order to justify the continuance of their neurotic patterns of behavior.[15] Many patients will present the anxiety of their fears of pain in the future even though at this time the actual perception of pain is minimal. The persistent complaint of pain as it occurs in many chronic illnesses has a phobic and compulsive character, and thus a significant psychologic meaning in terms of the personal and social life of the patient.[13] The role of psychic and emotional factors in various painful syndromes warrants the conclusion that one should approach each clinical pain problem not as being either somatic or psychic, but as a syndrome with elements of each involved in each case.[6] Each patient with severe intractable pain should be approached as an individual problem with the physician showing interest in the patient because the patient is not only *en rapport* with the physician, but he is also *en rapport* with himself. The success depends on the ability of the practitioner to prove his effectiveness to the patient. The sessions are time consuming for

the physician, but the results obtained provide a personal satisfaction of a job well done.

SUMMARY

Hypnosis as a state of hypersuggestibility has proved valuable in the relief of intractable pain. Therapy should be instituted before addiction to narcotics has occurred and should be considered before surgical procedures, such as prefrontal lobotomy and chordotomy, are performed. Success can be expected with this therapy although some patients will be unable to reach a deep trance. Suggestions in superficial states are not lasting, but the value of relaxation and interest of the physician in the individual problems is of value. Competency in hypnosis and the self-confidence of the physician tend to assure the patient of benefits to be derived with hypnotherapy. The dangers connected with the use of hypnosis are minimal in the hands of well-trained practitioners. Patients with hypnosis for intractable pain experience pain but do not let it reach the levels of consciousness.

2316 East Central St.
Wichita 2, Kansas

REFERENCES

1. Bonica, J. J.: *Management of Pain*, Lea & Febiger, Philadelphia, 1953.
2. Busse, E. W.: Application of hypnosis in medicine. South. M. J., *48:* 310, 1955.
3. Butler, B.: The use of hypnosis in the care of the cancer patient. Cancer, *7:* 1, 1954.
4. Capehart, M. P., Jr.: Present day methods concerned with the relief of intractable pain. J. Oklahoma M. A., *45:* 127, 1952.
5. Conn, J. H.: Therapeutic suggestion and hypnosis in every day practice. Maryland M. J., *7:* 592, 1958.
6. Cooper, I. S., and Braceland, F. J.: Psychosomatic aspects of pain. M. Clin. North America, *34:* 981, 1950.
7. Cooper, L. F., and Erickson, M. H.: *Time Distortion in Hypnosis*. Williams & Wilkins Company, Baltimore, 1954.
8. Erickson, M. H.: Hypnotic psychotherapy. M. Clin. North America, *32:* 571, 1948.
9. Heron, W. T.: Principles of hypnosis. South. M. J., *48:* 307, 1955.
10. Heron, W. T.: *Clinical Applications of Suggestion and Hypnosis*. Charles C Thomas, Publisher, Springfield, Ill., 1950.
11. Houde, R. W.: Pain and the patient with cancer. M. Clin. North America, *40:* 687, 1946.
12. Judovich, B., and Bates, W.: *Pain Syndromes*, Ed. 4. F. A. Davis Company, Philadelphia, 1953.
13. Kolb, L. C.: Psychiatric aspects of treatment for intractable pain in the phantom limb. M. Clin. North America, *34:* 1029, 1950.
14. LeCron, L. M., and Bordeaux, J.: *Hypnotism Today*. Grune & Stratton, Inc., New York, 1947.
15. Rosen, H.: The hypnotic and hypnotherapeutic control of severe pain. Am. J. Psychiat., *107:* 917, 1951.
16. Rosen, H.: *Hypnotherapy in Clinical Psychiatry*. Julian Press, Inc., New York, 1953.
17. Schneck, J. M.: *Hypnosis in Modern Medicine*. Charles C Thomas, Springfield, Ill., 1953.
18. Strauss, E. B.: Intractable pain. Brit. M. J., *2:* 411, 1949.
19. Weitzenhoffer, A. M.: *General Techniques of Hypnosis*. Grune & Stratton, Inc., New York, 1957.

THE SURGICAL SIGNIFICANCE OF RIB EXOSTOSES*

EDWARD R. HIPP, JR., M.D.

Charlotte, North Carolina

Despite adequate warning throughout the literature concerning the potential danger of malignant degeneration of originally very innocent appearing rib exostoses, some surgeons tend to dismiss these lesions summarily either by ignoring them or by treating them with inadequate excisional therapy. This is probably true because of the over-all rarity of these lesions and especially the rarity of such lesions in one man's personal experience. That the ecchondromatous lesions of the ribs have more malignant potential than similar lesions near the epiphyses of the long bones is a fact surrounded by established evidence.[2, 7, 28]

Even in the days before endotracheal anesthesia was commonplace, radical resection of the tumor with an adequate margin of rib and pleura, if necessary, was advocated. Hedblom,[10] in 1921, reviewed the literature and reported his experience at the Mayo Clinic. He was able to find approximately 213 cases in the literature up to that time and added approximately 48 from the Mayo Clinic. In 5 of his 10 cases the pleura was opened without any differential pressure apparatus, and there was no marked adverse reaction. Moreover, he stated that wide excision of the bony chest wall seemed to have no untoward effect in the remote postoperative period. Hedblom's statement,[10] "It seems evident from these considerations that the large majority of malignant growths of the chest wall may be expected to recur and metastasize after excision and that the so-called benign growths are liable to do so," is certainly corroborated by one of the case histories which will be related later in this paper.

Emphasis in the early literature is toward avoiding entrance into the pleural space. More recent experience, however, is that even in the small exostoses, comfortable surrounding lengths of rib, contiguous structures (whether they are rib or muscular chest wall), and an adequate margin of pleura and adherent underlying lung should be removed. There seems little excuse for homeopathic resections of these lesions or excisions directed toward avoidance of entrance into the pleural cavity.

The early literature[2, 3, 10] is replete with suggestions that trauma may play some part in the genesis of these rib lesions or in causing benign osteochondromas to undergo malignant degeneration. The postulates of Ewing must be fulfilled to substantiate such a cause and effect relationship. They are as follows: (1) The authenticity and sufficient importance of trauma must be established. (2) The previous integrity of the wounded part must be established. (3) A reasonable time relationship from 3 weeks to 3 years must have existed. (4) The identity of the injured area with the site of tumor must be established. (5) The tumor microscopically examined must be of a type that reasonably could be caused by trauma.

C. B. Morton,[19] in 1930, reviewed the literature and reported on a 63-year-old male with a large mass in the right side of the chest which had some association with trauma to the chest wall in the region of tumor development. He concluded that trauma could not bear more than a predisposing or secondary relationship to tumor formation because all trauma is not followed by tumor formation, and not all tumors are preceded by trauma. The relationship of tumor to trauma is now a rather moot question and apparently had no relationship in the 2 patients whose histories are herein presented.

Whether the ecchondromatous growths or exostoses are monostotic or polyostotic as in Ollier's disease, the same increased potential of malignant degeneration in the flat bones seems prominent. Of course, in the disease of multiple osteochondromatosis or ecchondromatosis, it is impractical to excise every rib on which there resides an osteochondroma. However, the lesions of the flat bones should be examined clinically at frequent intervals. Any growth tendency should be investigated by x-ray, and the area should be excised with an adequate margin of

* From the Department of Surgery, The Nalle Clinic, Charlotte, North Carolina.

Presented before the meeting of the Southeastern Surgical Congress, March 12, 1959.

normal tissue. Lichtenstein and Jaffe[18] describe a patient with multiple osteochondromatosis in which osteochondrosarcoma developed in the 6th rib.

Clinically, these patients present themselves with pain in the chest, which usually can be designated as being of chest wall origin by physical examination. Some of the lesions present themselves as masses on the ribs or are mistaken for breast masses or soft tissue tumors. In addition, the greatest source of discovery of these lesions is the survey chest x-ray. However, a great majority of the lesions of the ribs are overlooked on examination of the "mini films"; or they are noted in the report, and little significance is attached to the discovery of such an exostosis. This is most unfortunate because removal of such lesions is commensurate with our present day program of cancer prophylaxis.

The growth potential of the osteochondroma or exostosis of flat bones has been recognized by the pathologists for many years and probably precedes such awareness by the clinician. Lichtenstein and Jaffe[18] in 1943 stated, "Our own experience has taught us that a cartilage tumor is no longer to be regarded as benign if when viable, non-calcifying areas are examined, it shows even in scattered fields (1) many cells with plump nuclei, (2) more than an occasional cell with two such nuclei, and especially (3) giant cartilage cells with large single or multiple nuclei or with clumps of chromatin." They further state that many chondrosarcomas can be deduced to have arisen from benign lesions by their clinical course, their x-ray and pathologic findings. Moreover, they state that chondrosarcoma occasionally develops out of the cartilaginous cap of a solitary osteocartilaginous exostosis (so-called osteochondroma), perhaps more commonly of a flat bone or vertebra than a long bone. They state categorically that there is a tendency for the pathologist to "underdiagnose" such lesions.

Their criteria of benignity are as follows: (1) the component cartilage cells are fairly consistently rather small; (2) the vast majority of them have only one nucleus; (3) this nucleus tends strongly to be rather small and definitely not plump in relation to the cell as a whole; (4) such cells as binuclear are usually still small, have small and not plump nuclei and are found only occasionally and even then only in scattered fields; and (5) in particular, there are no large or giant cartilage cells with large single or double nuclei or with clumps of chromatin. On the other hand, they regard (1) many cells with plump nuclei, (2) more than an occasional cell with two such nuclei, and especially (3) giant cartilage cells with large single or multiple nuclei or with clumps of chromatin as definite indications of a malignant tumor. The possibility that rib tumors may be malignant from the start and not show their true disposition until they are greatly enlarged is entirely possible. Until this possibility is definite one way or the other, it is necessary to remove such lesions at an early stage.

Since the advice offered is to remove the tumor with a wide margin of normal tissue, one must necessarily discuss methods for closing defects occasioned by such radical excision. If the tumor involves only 1, 2, or 3 ribs, there is no problem as the chest musculature is adequate to cover any chest wall defect and also to obviate paradoxic respiration.

Should the excision, however, involve two-thirds or three-quarters of the supporting structure of the chest wall, then a number of considerations both mechanical and physiologic must be enumerated. The physiologic effect of paradoxic respiration can be lessened by the establishment of a tracheostomy, which reduces tidal volume enough to prevent hypoxia. Temporary stabilization of the chest wall by external splints or, more easily, either by incorporating the arm temporarily as a portion of the chest wall closure or using it to support the chest wall will decrease paradoxic motion of the chest wall and thereby prevent hypoxia. In removal of the large tumors of the chest wall there is rarely difficulty with finding some tissue to cover the defect. There is usually attenuated chest musculature and adequate, if not redundant, skin to cover the defect. Such coverage is entirely satisfactory as a temporary expedient and later may be found to be a definitive closure. Fascia lata or various metallic or plastic prostheses have been advocated as suitable and necessary for closure of chest wall defects. These it would seem are rarely needed at the primary procedure and may actually be detrimental to healing. Poppe and Berg[25] have advocated disarticulation of ribs superior and inferior to the result of excision and using these ribs by wedging or

kerfing them to bridge the defect. In such a closure the posterior end of the rib is left intact.

Recurrence of chest wall tumors should not always be regarded as an utterly hopeless situation. The recurrence should be circumscribed by surgical excision even if this involves removal of a portion of the diaphragm or entry into the peritoneal cavity or resection of portions of the lung. Harper[7] reports a huge chondrosarcoma which arose from the chest wall and extended into the thorax and abdomen. The tumor was removed by wide excision, and the patient was reported as showing a good short-term result.

The two following clinical summaries are presented to demonstrate extremities of the growth potential of these rib tumors.

CASE REPORTS

The first patient is an 11-year-old boy who was first seen on October 1, 1957, complaining of pain in the right anterior chest wall for a few weeks. This pain had no relation to position, exertion, and there had been no history of trauma to this region. Physical examination revealed a hard, nontender, nonmovable mass in the right anterior axillary line at the level of the 6th rib. X-ray revealed arising from the anterior portion of the 6th rib on the right a dumbbell-shaped bony extension consistent with an osteochondroma. This extended cephalad to impress the inferior aspect of the 5th rib. Tangential views of the chest revealed the tumor to have some intrathoracic extension. Complete physical examination on admission to Charlotte Memorial Hospital on August 13, 1958, revealed no abnormality other than a palpable mass 2 cm. in diameter, which was slightly tender, on the right chest wall at the level of the 6th rib (fig. 1).

FIG. 1. Osteochondroma in an 11-year-old boy arising from 6th right rib and impressing the inferior surface of the 5th rib.

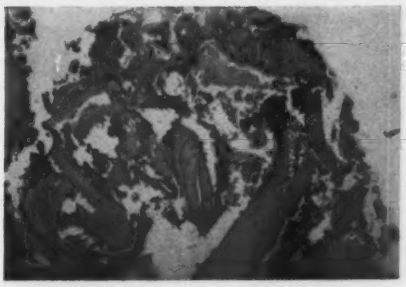

FIG. 2. Photomicrograph of osteochondroma in 11-year-old boy arising from the 6th right rib. Mature bone and cartilage are present.

On August 14, 1958, under endotracheal general anesthesia, a right lateral thoracotomy was performed, the chest being entered in the 6th interspace well anterior to the osteochondroma. At least 2 cm. on each side of the tumor of the 6th rib were removed, and a segment of the 5th rib, which was indented by the tumor, was also removed. A wide margin of pleura under these ribs was also excised. The chest wall defect was covered with the extra-thoracic muscles and skin. The postoperative recovery was uneventful, and the patient was discharged from the hospital approximately 1 week after surgery. Follow-up examination has been negative to date.

Pathologic study of the tumor revealed a 3-cm. excrescence on the segment of the 6th rib which was removed. There was an indentation in the bottom of the 5th rib. Microscopic examination revealed mature bone and cartilage consistent with an osteochondroma (fig. 2).

A complete bone survey was not done in this child because of the danger of radiation. All indications, however, are that this is a monostotic osteochondroma.

The next clinical summary is at the opposite end of the scale. This is briefly a presentation of a patient who first had a small osteochondroma of the rib which was removed incompletely, recurred, and grew for 13 years before the patient again would submit to surgical therapy.

The second patient is a 53-year-old colored man first seen at the Nalle Clinic on April 17, 1945, complaining of a lump in the right chest wall. An x-ray on this date revealed an osteochondroma of the 6th rib. On April 24, 1945, an attempt was made to excise the lesion, but the pleura was entered, and an incomplete excision was done. Pathologic study of the material removed revealed a chondroma (fig. 3). The pathologic interpre-

FIG. 3. Photomicrograph of a "chondroma" removed from the right 6th rib of a 53-year-old colored man on April 24, 1945. Very cellular chondroma with irregular cells, some showing plump nuclei. This was classified by the pathologist as benign microscopically, but he warned of the possibility of a malignant course of the disease.

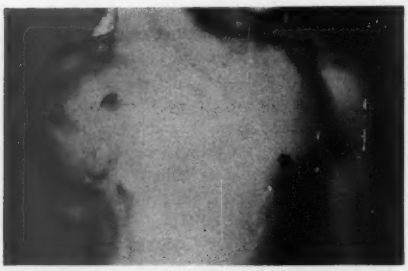

FIG. 4. Osteochondrosarcoma of the right chest wall present in a 53-year-old colored man for at least 13 years. (Previous attempt at resection 13 years before.)

tation given by Dr. Paul Kimmelstiel was, "The histologic examination of the material does not show evidence of malignancy. This, however, is not incompatible with a clinically malignant course of the disease. Other portions of the tumor may show a different histologic picture. Histologic diagnosis of benign chondroma should be taken with reservation."

The patient again returned to the Nalle Clinic on January 3, 1951, and presented himself with a very large recurrent tumor of the right chest wall. An x-ray on that date revealed, "There is a fairly smooth, rounded, irregular, calcified mass arising apparently from the 6th rib and destroying this rib. The mass measures approximately 14 x 11 centimeters and is situated in the lateral right chest. Metallic foreign bodies are seen in the left axilla. Impression: Osteochondroma or osteochondrosarcoma."

There is no mention in the chart history of the advice given to the patient nor what physical examination at that time revealed.

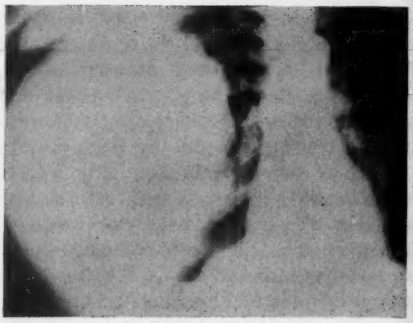

FIG. 5. Colored man, 53, with osteochondrosarcoma, right chest wall, involving ribs 4 through 9. Right lung is compressed by intrathoracic extension.

On July 8, 1958, he was again seen at the Nalle Clinic with a huge recurrent osteochondroma or osteochondrosarcoma of the right chest wall (fig. 4). This extended from the axilla to at least the 9th or 10th rib and extended anteriorly to the midclavicular line and about 5 cm. posterior to the posterior axillary line. It involved the tip of the scapula. The patient complained of pain, but his main complaint was that the mass appeared as some article underneath his shirt, which made his employer suspect him of stealing. There was some ulceration of the mass at its apical portion due to ischemia. The mass measured approximately 27 by 8 by 21 cm.

An x-ray taken on the same day revealed a tremendous tumor arising from the right chest wall involving ribs 4 through 8 (fig. 5). The x-ray interpretation was that the tumor had scattered cartilage and bone throughout it and that it had the appearance of a typical osteochondroma. The erosion of several ribs and possibly the inferior angle of the scapula from pressure effect alone was noted, but there was strong suspicion of this being osteochondrosarcoma. The right lung was seen to be compressed by this large tumor.

The patient entered Mercy Hospital on July 17, 1958. Generally, he presented the picture of a slightly emaciated colored man, appearing slightly younger than his stated age of 53 years, with a huge ulcerating tumor involving approximately the entire right chest wall laterally, extending posteriorly past the posterior axillary line and anteriorly to the midclavicular line (fig. 4). The mass was firmly fixed to the chest wall, but the skin over it could be moved over the tumor mass except for an area of about 3 cm. about the point of ulceration. There were no axillary nodes. There were no cervical nodes palpable. The blood pressure was 120/80. The heart was of normal size,

rate, and rhythm, and there were no murmurs. Abdominal examination revealed no evidence of enlargement of the liver, spleen or kidneys, nor were there any pathologic masses in the abdomen.

On July 18, 1958, the patient was taken to the operating room and placed in the right lateral decubitus position. Endotracheal anesthesia was induced. A long incision was made from the apex of the axilla to the edge of the rib cage with an ellipse of approximately 8 to 10 cm. in diameter in its center, avoiding with a wide margin the area of ulceration and fixation of skin to the tumor. A plane was developed just beneath the skin which left adherent to the tumor subcutaneous fat and chest wall musculature, including the greater portion of the latissimus dorsi. As dissection was developed anteriorly and posteriorly, more superficial portions of the pectoral muscle and lattisimus dorsi were left adherent to the skin flap. The chest wall was exposed, and the ribs were laid bare at least 4 to 5 cm. from the margin of the tumor attachment to the ribs at all points. The chest was entered anteriorly through the 2nd interspace, and the extent of the tumor into the thoracic cavity could be appreciated. The anterior ends of the ribs were severed close to the sternum, allowing elevation of the tumor. Ribs 4 through 9 were severed. The *en bloc* specimen could then be rotated posteriorly, and it was seen that the tumor involved the upper, middle, and lower lobes. A portion of the upper and middle lobe was removed with the tumor specimen, and the tumor was removed with a protective covering of pleura from the lower lobe (fig. 6). The posterior

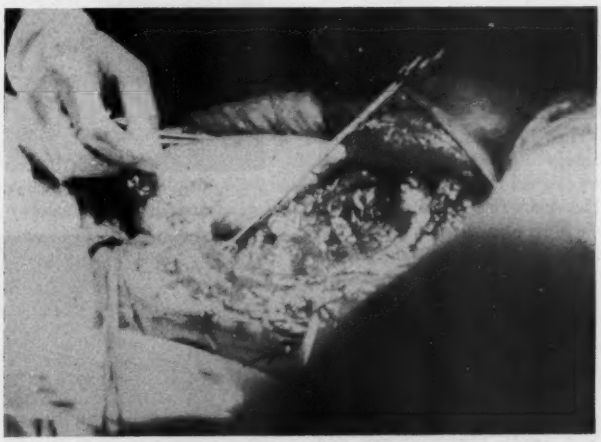

FIG. 6. Defect in chest wall after removal of osteochondrosarcoma involving 6 ribs. The right lower lobe can be seen, the fissure between middle and lower lobes being marked by sponge stick. Major portions of upper and middle lobes have been removed. (Nipple designated by *arrow*.)

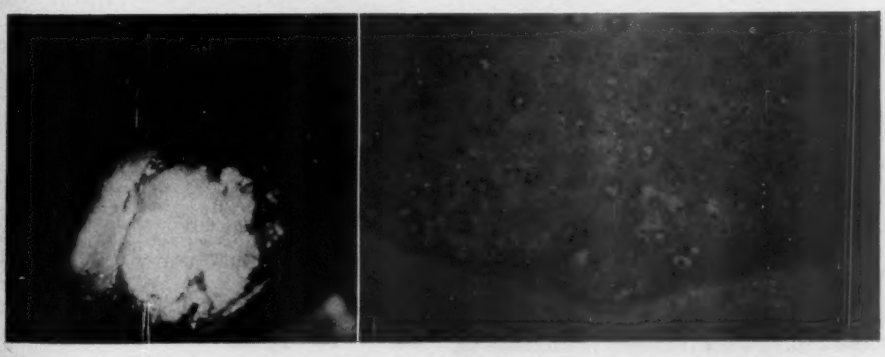

FIG. 7 (*left*). Tumor removed from right chest involving 6 ribs. Tumor weighed 12¾ lb.
FIG. 8 (*right*). Photomicrograph of osteochondrosarcoma showing increased cellularity, nuclear aberrations, and invasion of surrounding tissue.

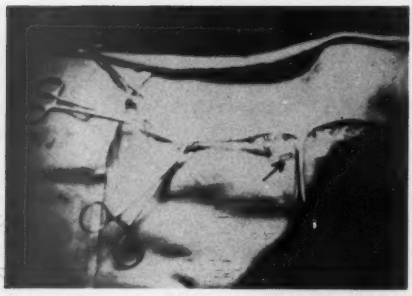

FIG. 9. Chest wall defect covered easily by attenuated chest musculature and skin (right lateral decubitus position).

segments of the 6 ribs involved in the tumor growth were then severed, and the specimen was removed. The specimen weighed 12¾ lb. in the fresh state (fig. 7). Pathologic study revealed osteochondrosarcoma (fig. 8).

The lung surfaces were seen to be free of any gross bleeding and air leaks following oversewing of the raw edges. The chest wall defect, which measured approximately 30 by 28 cm., was covered with the attenuated posterior and anterior thoracic muscles and the skin was approximated over the top of the muscle layers (fig. 9). No prosthetic appliance or fascia lata was used to further cover the defect in the chest wall. The patient received 6 units of blood during the procedure, and he tolerated the rather traumatic experience well.

Two intercostal tubes were placed and connected to underwater traps. The loose skin of the chest was fixed to the arm which was taped to the side to obviate as much as possible paradoxic motion of the chest wall. Tracheostomy was performed to further reduce the tidal air volume and to negate the effect of paradoxic motion in the chest wall as much as possible. The placement of the tracheostomy was certainly a life-saving measure in this particular situation.

Postoperatively, the patient did well (fig. 10). The chest wall closure healed by primary intention except for one point high in the axilla which drained seropurulent material for a few days. As the chest wall became more rigid and stable, the tracheostomy was gradually closed and finally removed without any untoward effect. In approximately 2 months the patient returned to light work. He developed no adduction contracture of the arm due to the long longitudinal incision (fig. 11). He complained of very little dyspnea except on extreme exertion. He has demonstrated at least 2 cm. variation in the skin of the chest wall, but this does not seem to affect his air exchange, particularly. In fact, when the skin is held forcibly, he complains that he is less easily able to breathe. The follow-up time is short, but he has demonstrated no evidence of recurrent tumor. He is now 8 months postoperative.

FIG. 10. Postoperative x-ray showing lack of reaction and good expansion of remaining lung.

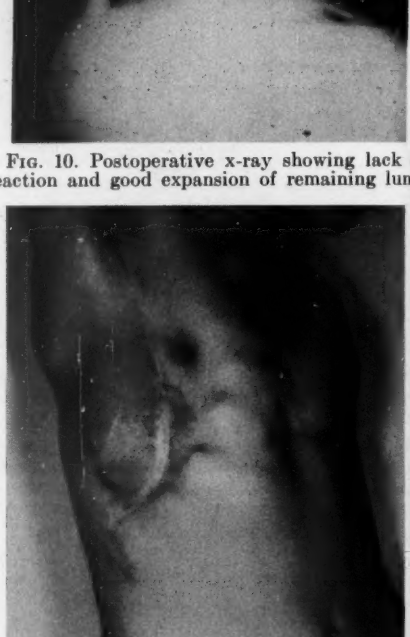

FIG. 11. Photograph of 53-year-old colored male 2 months postoperative. Note absence of scar contracture and adequate coverage of defect by skin and muscle.

CONCLUSIONS

In conclusion and summary the following statements seem to be in order.

1. Rib exostoses should not be summarily dismissed as being harmless lesions and unworthy of surgical attention when first discovered.

2. Osteochondromas of the ribs, either monostotic or polyostotic, should be regarded as *de novo* malignancies or potentially capable of malignant degeneration. They should be removed by adequate radical resection without regarding the pleura as other than a contiguous tissue which should be removed along with the specimen.

3. Chest wall defects created by excision of such lesions need not pose any great mechanical or physiologic problem. The removal of the tumor mass will result either in adequate or redundant musculature or skin which can be used to cover the defect either as a temporary expedient or as a definitive measure. Rarely should autogenous or prosthetic material be necessary.

4. The threat of paradoxic respiration can be managed either by incorporating the arm in the chest wall closure or by using it as a splint to the chest wall. The most important adjunct, however, is the use of the tracheostomy tube to facilitate suction, oxygen administration, and, more important, to diminish the tidal air volume.

5. One must remember that both the pathologist and the clinician tend to underdiagnose these lesions, and accordingly, the patient must be followed at regular intervals after surgery in order to determine immediately any evidence of recurrence.

6. Two more cases of primary rib tumor are added to the literature; one, a small osteochondroma of the right 6th rib and, second, a huge osteochondrosarcoma of the right chest wall involving 6 ribs.

The Nalle Clinic
Charlotte, North Carolina

REFERENCES

1. Auchincloss, H.: Osteochondroma of chest-wall. Ann. Surg., *100:* 399, 1934.
2. Blades, B., and Paul, J. S.: Chest wall tumors. Ann. Surg., *131:* 976, 1950.
3. Blake, J. M., and Bradford, J. K.: Primary fibrosarcoma of the chest wall following thoracic trauma. J. Thoracic Surg., *12:* 368, 1943.
4. Buckles, M. G., and Lawless, E. C.: Giant-cell tumor of ribs: case report and survey of literature. J. Thoracic Surg., *19:* 438, 1950.
5. Cohn, I.: A surgical clinic. Internat. Clin., *2:* 257, 1938.
6. Dorner, R. A., and Marcy, D. S.: Primary rib tumors: survey of literature and report of 7 additional cases. J. Thoracic Surg., *17:* 690, 1948.
7. Harper, F. R.: Benign chondromas of ribs. J. Thoracic Surg., *9:* 132, 1939.
8. Harper, F. R.: Huge chondrosarcoma arising from the chest wall and extending into the thorax and abdomen. J. Thoracic Surg., *11:* 446, 1942.
9. Harrington, S. W.: Surgical treatment of intrathoracic tumors and tumors of the chest wall. Arch. Surg., *14:* 406, 1927.
10. Hedblom, C. A.: Tumors of the bony chest wall. Arch. Surg., *3:* 56, 1921.
11. Hedblom, C. A.: Tumors of bony chest-wall; study of 22 personal and 78 collected cases since 1924. Ann. Surg., *98:* 528, 1933.
12. Heuer, G. J.: Thoracic tumors. Arch. Surg., *18:* 271, 1929.
13. Hochberg, L. A.: Primary tumors of the rib. A. M. A. Arch. Surg., *67:* 566, 1953.
14. Hochberg, L. A., and Crastnopol, P.: Tumors of the ribs. Dis. Chest., *28:* 406, 1955.
15. Jaffe, H. L.: Hereditary multiple exostoses. Arch. Path., *36:* 335, 1943.
16. Janes, R. M.: Primary tumors of ribs; report of 8 cases and a method of repair of the defect in the chest wall that follows their removal. J. Thoracic Surg., *9:* 145, 1939.
17. Kent, E. M., and Ashburn, F. S.: Ewing sarcoma of the rib. Am. J. Surg., *75:* 845, 1948.
18. Lichtenstein, L., and Jaffe, H. L.: Chondrosarcoma of bone. Am. J. Path., *19:* 553, 1943.
19. Morton, C. B.: Tumors of the bony thoracic wall associated with trauma. Am. J. Surg., *8:* 995, 1930.
20. Morton, J. J., and Mider, G. B.: Chondrosarcoma. Ann. Surg., *126:* 895, 1947.
21. O'Neal, L. W., and Ackerman, L. V.: Cartilaginous tumors of ribs and sternum. J. Thoracic Surg., *21:* 71, 1951.
22. Paget, S.: *The Surgery of the Chest.* J. Wright & Co., Bristol, 1896.
23. Pascuzzi, C. A., Dahlin, D. C., and Clagett, O. T.: Primary tumors of the ribs and sternum. Surg., Gynec. & Obst., *104:* 390, 1957.
24. Pendergrass, E. P., Lafferty, J. O., and Horn, R. C., Jr.: Osteogenic sarcoma and chondrosarcoma, with special reference to roentgen diagnosis. Am. J. Roentgenol. *54:* 234, 1945.
25. Poppe, J. K., and Berg, R.: Resection of chest wall tumors: report of 2 cases. Northwest Med., *47:* 119, 1948.
26. Samson, P. C., and Haight, C.: Giant-cell bone tumor of costal origin. J. A. M. A., *105:* 1020, 1935.
27. Sommer, G. N. J., Jr., and Major, R. C.: Neoplasms of the bony thoracic wall. Ann. Surg., *115:* 51, 1942.
28. Speed, K.: Tumors of the chest wall. Ann. Surg., *104:* 530, 1936.
29. Upshaw, J. E., McDonald, J. R., and Ghormley, R. K.: Extension of primary neoplasms of bone to bone marrow. Surg., Gynec. & Obst., *89:* 704, 1949.
30. Watson, W. L., and James, A. G.: Fascia lata grafts for chest wall defects. J. Thoracic Surg., *16:* 399, 1947.
31. Zinninger, M. M.: Tumors of the wall of the thorax. Ann. Surg., *92:* 1043, 1930.

MESENTERIC CYSTS

NORMAN F. SPRAGUE, JR., M.D.

Los Angeles, California

Cysts of the mesentery are rare and of historic interest. A Florentine anatomist, Antonio Benivieni,[6] to whom probably more than any other we owe the beginning study of gross pathology and pathologic anatomy,[30] may have encountered a mesenteric cyst, abscess or tumor during the latter years of the 15th century. In a little monograph[6] published in 1507, five years posthumously, consisting of a number of his most unusual cases, he describes a "callus" among the "mesaraic" (mesenteric) veins.

Then, according to the literature,[38] a few mesenteric cysts were described in the 17th and 18th centuries and in 1804 were classified by Portal,[32] physician to Louis XVIII of France. Warfield[41] says that in 1842, Rokitanski, the great pathologist who reputedly performed more than 30,000 autopsies, first described a chylous mesenteric cyst and attributed its origin to cystic degeneration of lymph nodes. Tillaux, in 1880, is said to have been the first to operate successfully upon a cystic mesenteric tumor. He was followed by Pean, who marsupialized one in 1883, while others were treated by several French and German surgeons.

In 1886, Augagneur[4] reported 90 cases of mesenteric tumor of which 18 were cystic. Arekion,[3] in 1891, referred to 91 cases, and the next year Braquehaye[9] in a very comprehensive report, reviewed the history of mesenteric cysts and added 23 cases for a total of 104. Moynihan,[28] in 1897, added 9 cases to the above and contributed some very pertinent observations to the study of this unusual condition. Then, in 1900, Dowd[13] in a classic paper recorded 32 additional cases making a total of 145, but felt that many of these were not reported sufficiently in detail to make them really intelligible. Numerous authors[5, 11, 17, 33] since that time have contributed to the literature and attempted to estimate the total number of cases reported. Higgins and Lloyd,[21] in 1924, felt that about 250 cases had been reported, chiefly by surgeons. Flynn,[16] in 1930, could find not more than 300 cases reported in the literature but felt the condition might be more common than generally believed. Burnett and his associates,[10] in 1950, found only 200 cases reported in any detail. Others,[24, 31, 36] have published estimates ranging from 300 to 600 as the total number of cases reported.

Vaughn and his co-workers[40] reviewed the literature in 1948 and stressed the rarity of this condition. They reported the third case of mesenteric cyst in 98 years and 750,000 admissions to Mercy Hospital in Chicago. Loeb,[26] in 1941, reported 1 case in 105,000 admissions to the Bronx Hospital in New York City. Block,[7] in 1948, reported only 1 case of chylous mesenteric cyst in over 160,000 admissions to the Jewish Hospital in Philadelphia, Pennsylvania. The largest recent series consists of 10 cases of isolated cysts in children reported by Gross,[18] in 1953. The following year Handelsman and Ravitch[20] published 4 cases, also in children. More recently, Collins and Holcomb[12] have reported 4 mesenteric cysts and 1 omental cyst, and suggest that the condition may not be so rare as previously assumed.

However, Alesen[2] reported the only case listed in the files of the Los Angeles County General Hospital from 1912 to 1929. A recent statistical review indicates that in the 30 years since then there have been about 14 cases of true mesenteric cysts in a total of 1,676,373 admissions. A statistical survey of the records at the Good Samaritan Hospital in Los Angeles fails to reveal any record under that diagnosis of a patient treated for a mesenteric cyst from January 1937 through December 1958 in 273,138 admissions. At the Harbor General Hospital there is no record of a true mesenteric cyst in 67,103 patients admitted from July 1952 through June 30, 1958. The 2 cases reported herein are the only cases of this kind seen at St. Vincent's Hospital in the past 30 years in 237,714 admissions.

Etiology

Much confusion exists in the literature relative to the genesis and classification of mesenteric cysts. Because the term has been used broadly it has at times included any cystic structure found within or in close relation to the mesentery.

Scientific accuracy was gained with the early grouping of cases, and in 1897, Moynihan[28] reviewed the anatomy of the mesentery and suggested a simple classification of the various forms of cyst which might be found therein; namely, (1) serous cysts, (2) chyle cysts, (3) hydatid cysts, (4) blood cysts, (5) dermoid cysts and (6) cystic malignant disease (cystic sarcoma).

This classification excluded all cases of cysts arising elsewhere and becoming mesenteric by later extension, particularly those from the ovary or pancreas. It also excluded various pathologic conditions of the mesenteric lymphatics other than cysts, such as lymph varices and cavernous lymphangioma. Moynihan also pointed out that serous cysts occur not only in the small bowel mesentery but also in the mesocolon, particularly, descending mesocolon. At the same time, he observed that unilocular cysts are much more common in the mesocolon, whereas multilocular cysts are seen more frequently in the mesentery proper.

Dowd,[12] in 1900, simplified this classification to include: (1) embryonic cysts (2) hydatid cysts and (3) cystic malignant disease. By separately classifying the parasitic and malignant cysts, he suggested all the remaining types originated as embryonic sequestrations. In this regard, one can readily visualize the ease with which a remnant from the primitive alimentary canal or a sequestration from the Wolffian body or embryonic genital ridge or a nest of primitive peritoneal cells could be included between the folds of the mesentery. Later, these cells could act as nuclei of cystic structures in the mesentery, mesocolon or omentum depending upon which particular developing area was invaded during their migration.

Dowd observed that chylous cysts were more common than any other variety of mesenteric cyst, and believed that they were preformed cysts of embryonic origin situated in such close relationship to the lacteals that chyle was effused into them. Similarly, the so-called sanguineous or blood cysts were, in effect, preformed cysts into which at some time hemorrhage had taken place. Likewise, serous cysts were felt to be of congenital origin with their fluid either exuded from blood vessels in the form of serum or secreted from the cells lining the cyst. Most of the serous cysts were noted to occur in the omentum, intestinal edge of the mesentery, mesocolon or retroperitoneal region, whereas the chyle cysts occurred in the mesentery proper.

Dowd also described certain "cysts with walls which suggest intestinal structure." Lewis and Thyng,[25] in 1907, observed the regular occurrence of intestinal diverticula in embryos of the guinea pig, rabbit and man. Guthrie and Wakefield,[19] in 1943, confirmed their findings and showed how these diverticula can extend into the mesentery and become pinched off. Later, these may be absorbed, or they may persist and form cysts. These presumably would be of the enteric type containing muscle elements in their wall and would in effect be cystic duplications of the bowel. Gross[18] has emphasized that enteric cysts are thick walled and lie immediately adjacent to the bowel, with their musculature so intimately associated that they cannot readily be removed without impairing the blood supply to the intestinal segment. In contrast, true mesenteric cysts are thin walled and have no muscular coat or mucosal lining.

In spite of the many excellent papers published since the turn of the century surprisingly few new facts have been added to our knowledge of the genesis of these cysts. Numerous authors[15, 19, 29, 34, 35, 37, 39] have amplified the classification of mesenteric cysts of embryonic origin, but the basic theory has not been changed and has recently been restated by Ackerman.[1]

Pathology

It has been noted that mesenteric cysts are ordinarily single, but occasionally may be multiple. They may be unilocular or multilocular. In size, they vary from those just large enough to recognize to huge cysts filling the abdomen. Their contents, in most instances, consist of clear serous fluid, but may be a milky chyle, mucinous or serosanguineous in nature. Gross[18] has noted that all cysts in his series with chylous fluid were removed from the mesentery of the upper ileum or jejunum where material draining from the intestinal tract presumably contained a high percentage of fat. Those cysts in the mesentery proper are often flabby and may have a dumbbell shape projecting from the surface of the mesentery on both sides of the intestine. Those removed from the mesocolon tend to be unilocular and may be filled with clear serous fluid to an appreciable degree of tenseness.

True mesenteric cysts all lie between the

peritoneal leaves of the mesentery, with the majority located in the jejunum or ileum and the remainder in the mesocolon. Their wall is ordinarily thin, rarely more than 2 mm. in thickness, and consists primarily of connective tissue. Histologically, it may have such specialized cytologic detail as to suggest the derivation of the cyst.[24]

To summarize, the term mesenteric cyst merely indicates a cyst within the mesentery and, therefore, may include not one but a variety of cystic formations. Of these, the traumatic, parasitic, inflammatory, neoplastic and dermoid cysts are either readily classified or belong in a characteristic group of their own. The remaining mesenteric cysts are probably of developmental origin and might be classed on an etiologic basis as follows: (1) enteric, (2) urogenital, (3) lymphatic and (4) celomic.

Enteric cysts characteristically have thick walls including muscle and mucosal elements (which as the cyst becomes larger may be replaced with fibrous tissue) and lie close to the adjacent intestine.

Urogenital cysts are derived from rudiments of the embryonic urogenital system. They are generally simple unilocular cavities lined with columnar cells and containing clear fluid. Ordinarily they are located in the retroperitoneal area and not between the leaves of the mesentery. It must be realized, however, that structures arising in the retroperitoneal area may force their way between the leaves of the mesentery, and conversely true mesenteric cysts as they enlarge may become partially retroperitoneal.

Lymphatic cysts are probably for the most part lymphangioms of congenital origin. They tend to occur in younger individuals, and their contents are frequently clear although they may be filled with chyle from adjacent lacteals or blood from hemorrhage. Their wall is composed of fibrous and elastic tissue usually lined with a flat endothelium.

Celomic or mesothelial cysts may be derived from isolated nests of primitive celomic or peritoneal cells which form a cyst in their attempt to duplicate the celomic or peritoneal cavity.[23] Presumably, in the mesentery proper they could be entrapped in one of the numerous folds during the developmental process, whereas, in the mesocolon, it has been stated,[22] their presence may be associated with a failure of coalescence of the 2 posterior layers of parietal peritoneum during the 3rd stage of intestinal rotation. The lining of these cysts consists of mesothelial cells of a low cuboidal nature, lower than the typically columnar cells of urogenital origin and higher than the frequently flattened-out cells of endothelial origin. This type of cyst would appear to be more common in the mesocolon.

Diagnosis

Mesenteric cysts have been reported in patients of all ages from newborn infants to octogenarians[3] but are now probably diagnosed more frequently in childhood or early adulthood.[27] There are no signs or symptoms pathognomonic of this condition. The initial complaint may be only a painless progressive enlargement of the abdomen. The great majority of cases, however, are associated with pain[10] related rather directly to the size and location of the cyst. In some instances, this may be only a vague discomfort due to the mechanical effect of the mass. More commonly, the symptoms of a low grade intestinal obstruction are present with intermittent episodes of cramping, mild to moderate abdominal pain. Anorexia, nausea and vomiting as well as alternating constipation and diarrhea may be associated. More serious complications occasionally reported have been acute intestinal obstruction, volvulus, intussusception, hemorrhage into a cyst, acute inflammatory changes and peritonitis due to spontaneous rupture of a cyst.[14]

Some mesenteric cysts are so soft they cannot be felt through the abdominal wall. In most instances, however, palpation will disclose a smooth, rounded, nontender cystic tumor which is characteristically quite mobile, especially in the transverse direction. Moynihan[28] indicates that the most prominent part of the tumor is generally found toward the umbilicus with some possibility of rotation about a central axis. He states that Tillaux has described a zone of resonance around and a belt of resonance across these cysts.

Roentgenograms may reveal a homogeneous density displacing stomach, intestines or colon into other parts of the abdomen. Barium studies can rule out involvement of the gastrointestinal tract. Pyelography may reveal renal or ureteral displacement while eliminating hydronephrosis as a possible diagnosis.

Treatment

The treatment of a mesenteric cyst is surgical, and the procedure of choice is enucleation with

complete removal of the cyst. If this is not possible without impairing the vascular supply to the adjacent bowel, the cystic mass may be excised along with a portion of the intestine and continuity re-established by anastomosis. In the rare instance in which for some reason either of the above cannot be accomplished, marsupialization is said to have yielded satisfactory results. It is possible that some form of internal drainage procedure might be of use under certain conditions. Aspiration as a method of treatment should certainly be avoided.

CASE REPORTS

Case 1. E. W., a 44-year-old white man, was seen May 22, 1958, complaining of a "sense of uneasiness" in the left lower quadrant (LLQ) of his abdomen. This was associated with loose bowel movements and mild bloating. The patient had first noted the onset of these symptoms in Acapulco, Mexico, about 8 weeks previously, following a yacht race. Several of his companions on this trip had developed jaundice. The patient's symptoms were mild, and the abdominal discomfort was not accompanied by diarrhea, vomiting, fever, urinary symptoms or weight loss. There was no history of trauma to the abdomen.

In 1954, the patient complained of transitory epigastric discomfort associated with mild constipation and chest pain. Abdominal examination by two internists and by the author at that time was completely negative. Gastrointestinal and barium enema x-rays revealed no evidence of intrinsic or extrinsic pathology. Past history was otherwise negative except for an appendectomy performed some years before.

The family history was essentially noncontributory. One brother had died of acute monocytic leukemia. There were no known congenital defects in the patient or his family.

The pertinent findings on physical examination were restricted to the abdomen. It was soft throughout with an easily palpable, apparently cystic mass the size of a large grapefruit on the left side just above the level of the umbilicus. The mass was somewhat movable anteriorly but well fixed posteriorly. It was not tender, but on movement caused a sensation of mild distress in the LLQ. Peristalsis was normal.

Laboratory examinations revealed red blood cells (RBC), 4,850,000; hemoglobin (Hgb.), 13.2 gm. (86 per cent); color index, 0.89; white blood cells (WBC), 6350 with a normal differential

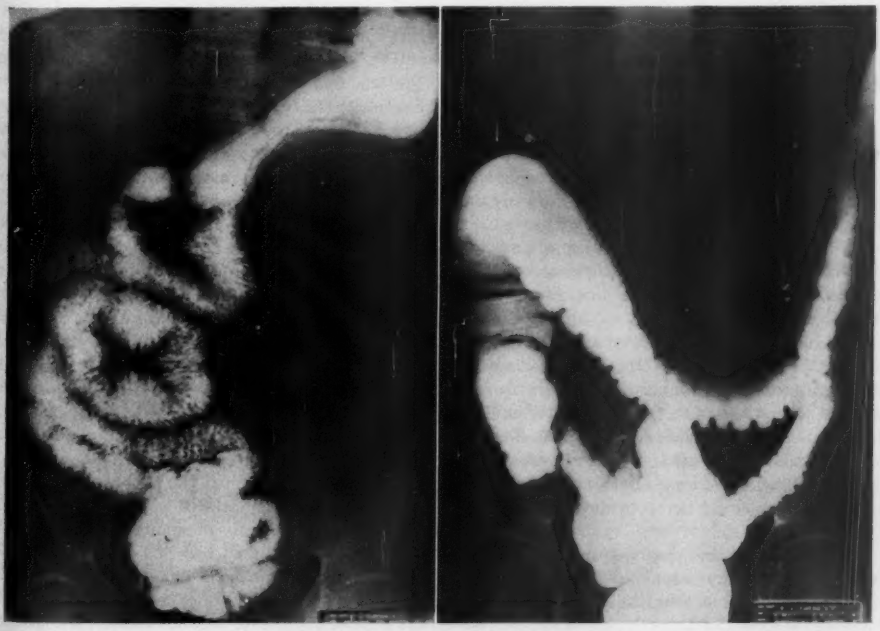

FIG. 1 (*left*). Gastrointestinal series demonstrating the deformity of stomach and displacement of small intestine by cyst.
FIG. 2 (*right*). Barium enema showing displacement of colon by cyst.

FIG. 3. Intravenous pyelogram demonstrating elevation, distortion and compression of left renal system.

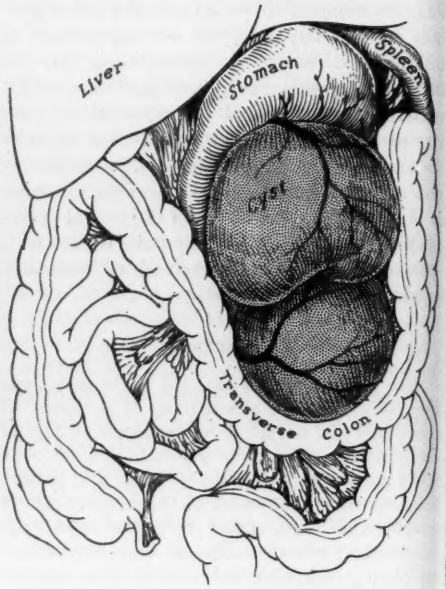

FIG. 4. Drawing to illustrate anatomical location of cyst.

urine negative except for a faint trace of albumin, a rare WBC and RBC and a very occasional finely and coarsely granular cast. Fasting blood sugar was 104 mg. and nonprotein nitrogen (NPN), 33 mg. per 100 cc. of blood. Amylase was 68 units (Somogyi method-normal 40–110). Quick prothrombin concentration was 33 per cent, but, Bromsulphalein, thymol turbidity, cephalin-cholesterol flocculation, total serum proteins and albumin-globulin (A/G) ratio were within normal limits. Bleeding and clotting times, platelets and reticulocytes were normal. Following administration of vitamin K, the quick prothrombin time gradually rose to 100 per cent.

Roentgenograms revealed a large posterior, apparently retroperitoneal mass displacing the greater curvature of the stomach anteriorly and cephalad, the jejunum to the right of the midline (fig. 1), and the colon inferiorly (fig. 2). The left renal system was elevated, distorted and slightly compressed (fig. 3). It was believed that a separate splenic shadow could be seen, deformed and rotated by the mass.

It was felt that the mass was probably a pancreatic cyst, with mesenteric cyst to be ruled out, and the patient was admitted to St. Vincent's Hospital for an exploratory laparotomy.

The abdomen was opened through a mid-transverse incision and the tumor found to be a large, relatively thin-walled cyst occupying the greater portion of the left abdominal cavity (fig. 4). It was encompassed by the lower portion of the transverse mesocolon and extended from Treitz's ligament superiorly to the pelvic brim inferiorly (fig. 5). The cystic mass was fixed posteriorly by a broad vascular pedicle arising from the inferior mesenteric artery (fig. 6). A smaller vascular pedicle originated from the middle colic vessels and spread over the anterior aspect of the cyst. It was relatively easy, by using meticulous dissection, to mobilize the cystic mass and remove it intact (fig. 7). There was no association between the cyst and the pancreas. The ureter was exposed, but undisturbed. In the region of Treitz's ligament it was necessary to shell part of the cyst out of the serosal wall of the 3rd portion of the duodenum. The patient tolerated the procedure well, and his convalescence was uneventful. A year after the surgical procedure, he was completely asymptomatic.

Kahler[22] has described the pathology as follows: "The specimen consists of a large cyst from the mesentery of the transverse and descending colon so that the cyst wall touched the serosa of the splenic flexure and toward the midline that of the third portion of the duodenum. The cyst measures 25 x 20 x 10 cm. It has an ovoid but indented outline being somewhat reniform with the vascular pedicle entering a deep cleft near the center of the

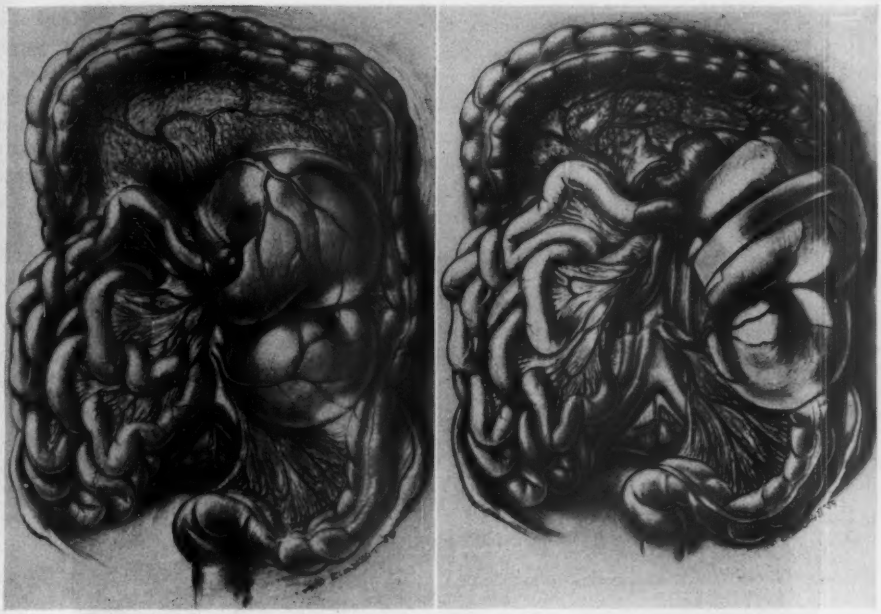

FIG. 5 (*left*). Drawing of cyst before enucleation.
FIG. 6 (*right*). Drawing of cyst during surgical removal.

lesion. When it is opened it proves to be a unilocular cyst with a thin, translucent wall and with a perfectly smooth lining membrane. It is filled with clear, limpid watery fluid.

"The cyst is lined by one to two layers of cells which at times are so flat as to resemble endothelium. At other times they are cuboidal or elongated in outline like mesothelium. This latter appearance occurs principally where the cells are in two layers. The cells are always flat. They are never raised in any papillary projections. They rest directly upon the wall which is composed of concentrically arranged adult, fibrous tissue cells. There is no muscle in the wall except that of the blood vessels. There is an occasional focus of lymphocytes in the wall, and there is a large focus of lymphocytes in the vascular pedicle."

Diagnosis: Celomic (mesothelial) cyst.

Case 2. C. M., a 25-year-old white woman, private patient of Dr. Henry Thomas, was admitted to St. Vincent's Hospital on September 28, 1949. The patient was a multipara about 6 weeks pregnant. A slightly tender, freely movable large round cystic tumor was easily palpable in her abdomen. This had been completely asymptomatic and was found on routine physical examination. The patient's only complaints were those relating to some associated bilateral varicose veins.

Past history was essentially negative; family history was noncontributory. Laboratory work show RBC, 4,430,000; Hgb., 11.3 gm. (75 per cent); WBC, 5000 with normal differential. Urine was negative except for rare WBC and Kline negative.

A presumptive diagnosis of ovarian cyst was made and the patient taken to surgery by Dr. Thomas. The abdomen was opened through a low midline incision. A large cystic mass was found in the mesentery of the jejunum. It was filled with clear fluid, and there was no evidence of inflammation about it. Abdominal findings were otherwise negative except for those compatible with an early pregnancy. The cyst then was carefully dissected out except for a very small portion of the wall which was so adherent to the mesenteric vessels that it was allowed to remain. Postoperative course and convalescence were uneventful. There has been no known recurrence in the 10 years since surgery.

The pathology was reported by Kahler[23] as follows: "The specimen consists of a cystic tumor removed from the mesentery of the jejunum. It measures 17 x 15 x 5 cm. and is multilocular, the largest locule being 15 x 15 x 5 cm. and the smallest 3 cm. in diameter. All are similar in appearance

FIG. 7. Gross specimen of cyst

in that they have a very thin translucent wall, a perfectly smooth, practically invisible lining membrane and are filled with clear limpid watery fluid.

The cystic spaces are lined by a single layer of flattened cells resembling vascular endothelium. These rest upon a thin membrane of connective tissue which contains no muscle elements. There are scattered lymphocytes in the cyst walls."

Diagnosis: Lymphangioma of mesentery (cystic hygroma).

SUMMARY

Mesenteric cysts are rare and of interest historically. Their etiology, classification and pathology have not as yet been finally or clearly delineated, although they are almost certainly of congenital origin. The symptomatology of these

cysts is related to their size and location in the abdomen, and they are frequently associated with some degree of intestinal compression or obstruction. Their treatment is surgical and currently the mortality should be low.

Review of the records of 4 hospitals in this vicinity confirms the incidence of true mesenteric cysts to be less than 1 per 100,000 admissions. Two typical cases of mesenteric cyst are reported. One is a unilocular celomic or mesothelial cyst originating in the transverse mesocolon. The other is a multilocular lymphatic or chylous cyst occurring in the mesentery proper. A simple classification of mesenteric cysts of developmental origin, based on their etiology, is suggested.

Norman F. Sprague, Jr., M.D.
2010 Wilshire Boulevard,
Los Angeles 57, California

REFERENCES

1. Ackerman, L. V.: Tumors of the retroperitoneum, mesentery and peritoneum. Armed Forces Institute of Pathology, Sec. 6, fasc. *23-24:* 87, 1954.
2. Alesen, L. A.: Mesenteric chylous cysts. California & West. Med., *30:* 261, 1929.
3. Arékion, H. F.: Étude sur les kystes du mésentère. These. Paris, 1891. Quoted by Dowd.[13]
4. Augagneur, V.: Tumeurs du mésentère, these d'agregation. Paris, 1886. Quoted by Dowd, Delahaye and Lecrosnier.
5. Benedict, A. L.: Bibliography of chylous cysts of the mesentery. Surg., Gynec. & Obst., *16:* 606, 1913.
6. Benivieni, A.: *De Abditis Nonnullis Ac Mirandis Marborum et Sanatiorium Causis*, Florentine, 1507. *Hidden Causes of Disease*, translated by Charles Singer. Charles C Thomas, Springfield, Ill., 1954.
7. Block, F. B.: Chylous mesenteric cyst. Ann. Surg., *128:* 158, 1948.
8. Blackmun, R. L.: Mesenteric cysts. Am. J. Surg., *77:* 371, 1949.
9. Braquehaye, J.: Des kystes du mésentère. Arch. Gén. de Méd., *2:* 291, 572, 1892. Quoted by Moynihan.
10. Burnett, W. E., Rosemond, G. P., and Bucher, R. M.: Mesenteric cysts. Arch. Surg., *60:* 699, 1950.
11. Carter, R. M.: Cysts of the mesentery. Surg., Gynec. & Obst., *33:* 554, 1921.
12. Collins, H. A., and Holcomb, G. W.: Mesenteric and omental cysts. Am. Surgeon, *24:* 763, 1958.
13. Dowd, C. N.: Mesenteric cysts. Ann. Surg., *32:* 515, 1900.
14. Eliason, E. L., and North, J. P.: Perforated chylous cyst of the mesentery. Ann. Surg., *101:* 1452, 1935.
15. Ewing, J.: *Neoplastic Diseases*, Ed. 4. W. B. Saunders Company, Philadelphia, 1940.
16. Flynn, C. W.: Mesenteric cysts with report of case of cystic lymphangioma. Ann. Surg., *91:* 505, 1930.
17. Friend, E.: Mesenteric chyle cysts. Surg., Gynec. & Obst., *15:* 1, 1912.
18. Gross, R. E.: *The Surgery of Infancy and Childhood.* W. B. Saunders Company, Philadelphia, 1953.
19. Guthrie, R. F., and Wakefield, E. G.: Mesenteric cysts. Proc. Staff Meet. Mayo Clin., *18:* 52, 1943.
20. Handelsman, J. C., and Ravitch, M. M.: Chylous cysts of the mesentery in children. Ann. Surg., *140:* 185, 1954.
21. Higgins, T. T., and Lloyd, E. I.: Mesenteric cysts with report of two cases. Brit. J. Surg., *12:* 95, 1924.
22. Illingsworth, C. F. W., and Dick, B. M.: *A Text Book of Surgical Pathology*, Ed. 7. Little, Brown & Company, Boston, 1956.
23. Kahler, J. E.: Personal communication.
24. Keesey, W. E.: Cysts of the mesentery. Illinois M. J., *73:* 333, 1938.
25. Lewis, F. T., and Thyng, F. W.: The regular occurrence of intestinal diverticula in embryos of the pig, rabbit and man. Am. J. Anat., *7:* 505, 1907.
26. Loeb, M. J.: Mesenteric cysts: review of literature, genesis and classification. New York J. Med., *41:* 1564, 1941.
27. Moore, T. C.: Congenital cysts of the mesentery. Ann. Surg., *145:* 428, 1957.
28. Moynihan, B. G. A.: Mesenteric cysts. Ann. Surg., *26:* 1, 1897.
29. Niosi, F.: Die Mesenterialcysten Embryonalen Ursprungs nebst einigen bem erkungen zur entwicklungs geschichte der nebennieren-rindensubstanz sowie zur frage des chorionepithelionig. Virchows Arch. path. Anat., *190:* 217, 1907.
30. Park, R.: *An Epitome of the History of Medicine*, Ed. 2. F. A. Davis Company, Philadelphia, 1899.
31. Parsons, E. O.: True proliferating cystic lymphangioma of the mesentery. Ann. Surg., *103:* 595, 1936.
32. Portal, A.: *Cours d'Anatomie Médicale, ou Éléments de l'Anatomie de l'Homme.* Baudouin, Paris, 1804. Quoted by Dowd.[13]
33. Porter, M. F.: Chylous cysts of the mesentery. Ann. Surg., *43:* 380, 1906.
34. Robbins, S. L.: *Textbook of Pathology*, Ed. 1. W. B. Saunders Company, Philadelphia, 1957.
35. Roller, C. S.: Mesenteric cysts. Surg., Gynec. & Obst., *60:* 1128, 1935.
36. Slocum, M. A.: Surgical treatment of chylous mesenteric cyst by marsupialization. Am. J. Surg., *41:* 464, 1938.
37. Snyder, J. W.: Mesenterie cysts. South. Surgeon, *8:* 240, 1939.
38. Swartley, W. B.: Mesenteric cysts. Ann. Surg., *85:* 886, 1927.
39. Thompson, G. C. V., and Chambers, C. H.: Chylangioma of the mesentery, with report of a case and a brief discussion of mesenteric cysts. M. J. Australia, *1:* 210, 1946.
40. Vaughn, A. M., Lees, W. M., and Henry, J. W.: Mesenteric cysts. Surgery, *25:* 306, 1948.
41. Warfield, J. O., Jr.: A study of mesenteric cysts. Ann. Surg., *96:* 329, 1932.

ANTERIOR EXCISION OF RUPTURED CERVICAL INTERVERTEBRAL DISC AND INTERBODY FUSION (CLOWARD)*

RALPH M. STUCK, M.D., ROBERT F. HALL, M.D., AND E. HOWARD FRALICK, M.D.

Denver, Colorado

The increasing speed of modern transportation has resulted in a marked increase in accidents which shock and damage the human body. Collisions at these increased speeds result in damage to the bodily structures that are most vulnerable either by their inherent nature or by the situation in which they are placed. Most parts of the body if protected by crash pad, safety belt, head rest or other means of deceleration may escape serious damage. But very few means of transportation provide protection for the cervical spine. As a result, neck injury is rapidly becoming a major problem.

The neck may be damaged in other ways than by automobile accident. For instance, a person may slip and fall, striking his head in such a fashion as to wrench his neck. He may receive a blow that compresses his neck or may sustain a direct blow to the neck. Injury may also occur to the cervical structure from repeated attritions, as in boxing or jolting.

As a result of such injury, the soft tissues of the neck, the cervical spine and the cervical intervertebral discs may be contused, lacerated, fractured or ruptured. The stress, often applied in a whip-cracker or shearing-tearing fashion, may result in the stretching and tearing of ligaments, muscles and other soft tissue structures; the stretching, tearing or crushing of nerve structures; the fracture and dislocation of bony structures; and the crushing, fracturing and dislocation of disc structures.

The location and extent of the damage to the neck structures can be determined in part by the clinical symptoms and the objective physical findings. Headache and shoulder pain, although usually indicating a general effect upon these structures, may assist in identifying the exact neck structures injured and the extent of their injury. Pain may be in the neck and the back of the head and may extend forward behind the eyes; it may be in the region of the scapula, in the pectoral region or in the shoulder, arm, forearm or hand. Numbness and tingling may be experienced in the arm, forearm, hand or fingers. Weakness may be felt in the muscles of the shoulder girdle, the arm, the forearm or the intrinsic muscles of the hand. Tenderness may be found along the cervical spine, along the vertebral border of the scapulae and in the muscles which are also painful. Weakness and wasting may be seen in the muscles of the shoulder girdle, the arm, the forearm and the hand. Sensory changes may be difficult to elicit, but they should be searched for diligently as they may be indicative of nerve root injury. Reflex changes, too, may indicate nerve root involvement. In this survey we have found Keegan's plan of distribution of cervical nerve roots to be most accurate.

In addition to the physical signs, x-rays may be of extreme value in establishing a diagnosis in neck injury. Our experience with cases of neck injury has led us to conclude that the cervical intervertebral discs are damaged much more often than has been believed in the past and that many patients with neck injury and a diagnosis of neck sprain actually have one or more symptomatic ruptured cervical intervertebral discs that require excision and spinal fusion. By using the discogram,[2] a relatively new x-ray procedure in the cervical region, we have demonstrated damaged discs in most neck injured patients whose symptoms have not subsided under conservative treatment in a period of three months. Moreover, we have found that anterior disc excision with nerve root compression and interbody fusion brings relief of these symptoms.

Our experience has led us to conclude that the presence of the neurologic and clinical pattern of symptoms commonly attributed to injured cervical discs, often called the cervical syndrome, is not necessary to establish the diagnosis of ruptured cervical intervertebral disc. Routinely we examine and record the complaints and physical findings in these patients. We also endeavor to obtain x-rays of the cervical spine

* Presented at the Eleventh Annual Meeting of the Southwestern Surgical Congress, Denver, Colorado, March 30, 1959.

which include anteroposterior, lateral, oblique, flexion and extension views. We no longer find myelography essential unless a spinal cord tumor is suspected.

Cervical discography has proved to be the most significant test in determining cervical disc injury. Through its use, the disc or discs involved can be determined accurately, the extent of the damage can be estimated, and the clinical symptoms can be reproduced during the test, a fact that further accurately localizes the lesion. A patient with an injured neck should be treated conservatively for a period of three months. At the end of this time if his symptoms are still disabling, we believe a discogram should be performed. If the discogram reveals disc injury and if during this test his symptoms are aggravated, he should be operated upon.

Until recently almost all cervical disc surgery was performed by posterior laminectomy. Often cases were classified as to whether or not bony reaction occurred and whether the disc was soft or hard, laterally or medially displaced. Various modifications of posterior laminectomy were used depending on the location and character of the disc. Some medical writers have reported a relatively large series of cases. Posterior cervical laminectomy is technically difficult and in many instances hazardous. It involves deep muscle splitting and partial or complete excision of laminal arches. It is often accompanied by excessive epidural venous bleeding. Since in most instances the spinal cord must be handled, additional neurologic injury can result. The convalescence is long, and permanent partial disability may be marked. A few cases have been permanently totally disabled. Cleveland[1] states that ". . . surgical treatment for the cervical syndrome is generally a last resort and should be used only after non-surgical treatment fails." Consequently, most surgeons who have done posterior cervical laminectomies continue to search for a better method.

About 10 years ago, interest was first shown in an anterior surgical approach to the cervical disc problem, but no definite procedural plan was developed. Then in 1955 Smith and Robinson[5] reported an anterior approach that achieved quite favorable results.

Cloward[3] of Honolulu, Hawaii, after surveying the problem of ruptured cervical disc and studying the anterior surgical approach to the cervical vertebrae, developed the method which we are using. The incision is made anteriorly in the neck, and dissection is continued laterally to the trachea and esophagus and medially to the sternomastoid muscle and the structures contained in the carotid sheath. The anterior surface of the vertebral column is exposed. The intervertebral disc to be operated upon is again identified by x-ray and is excised by sharp dissection and by means of a coring drill. The nerve roots in the nerve foramina are decompressed by removing the medial wall of the bony foramina along with the encroaching osteophytes. Finally a plug of bone is cut from the ilium and inserted between the vertebrae (fig. 1). Distraction of the vertebra and interbody fusion thus result.

Cloward states, "Technically it is possible to remove completely both soft-tissue and bony elements encroaching upon the nerve roots or the spinal cord a great deal more satisfactorily than has ever been possible by the posterior approach. The dangers of injury to the spinal cord and nerve roots in the posterior operation, usually by hemorrhage and/or retraction, are almost

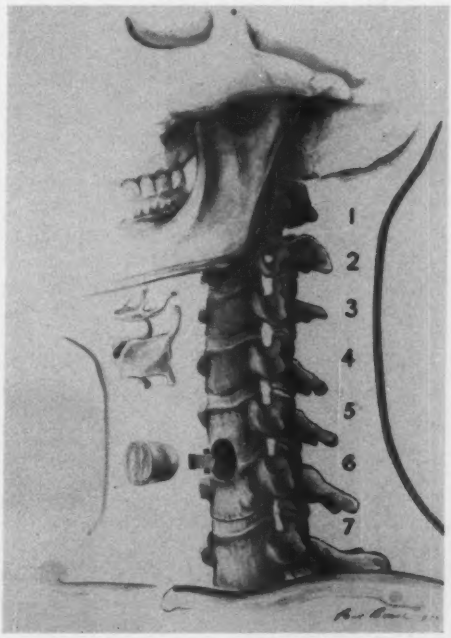

FIG. 1. Fusion plug being placed between vertebral bodies

entirely eliminated. Finally, the most important contribution to this treatment of degenerated cervical disk (cervical spondylosis) by this new procedure is the fact that the vertebral bodies are firmly fused together in a very short period of time. This arrests the degenerative process at the joint, which, if permitted to continue, may accumulate large osteophytes on the margin of the vertebra with encroachment upon the spinal canal and possibly result in chronic irreparable damage to the spinal canal and/or nerve root."

In the cases we are reporting, we have used exclusively the technique Cloward described and demonstrated to us. We are convinced that the operative procedure of choice for disabled cases of ruptured cervical intervertebral disc identified by discography is anterior excision of the cervical disc, decompression of the nerve roots and interbody fusion.

CASE REPORTS

Case 1 (*RPB-RFH 7703-A*). This 21-year-old white woman was injured on March 11, 1958, when her car was hit from behind by another car, and her neck was forcibly jerked backward. She was seen within an hour after her initial injury and was immediately hospitalized after check x-rays had been obtained. She was treated conservatively with bed rest, head and neck traction, ultrasonic therapy, antispasmodics, sedatives and analgesics. She complained chiefly of pain in the neck with radiation into the right posterior occipital area. She was hospitalized for 11 days and on discharge was sent home with a cervical collar, continuation of head and neck traction and other palliation.

Her initial x-rays revealed flattening of the normal cervical curve but no other evidence of any true bone or joint pathologic change. She was then treated symptomatically with conservative therapy consisting of head and neck traction, cervical collar, antispasmodics and drugs. On April 19, 1958, she was given intermittent head and neck traction. This gave her only temporary relief of neck pain. In addition she had multiple field blocks to the cervical spine at the base of the skull which also gave her only temporary relief.

The patient was admitted to the hospital on

FIG. 2 (*left*). Lateral view, cervical spine. Discogram at C5-6 permitted injection of 0.3 cc. of contrast media. Configuration is normal and no symptoms were produced.

FIG. 3 (*right*). Anteroposterior view, cervical spine. Discogram at C6-7 permitted injection of 0.9 cc. of contrast media. Configuration is abnormal and severe scapulotrapezius pain was produced on left at the moment of the injection.

June 6, 1958, having had approximately 3 months of conservative therapy. Discography was performed under general anesthesia on June 7, 1958, on the operating table and revealed a ruptured C4-5 disc. The C4-5 intervertebral disc was then excised, and an anterior intervertebral spinal fusion was performed.

Postoperatively, the patient did quite well. Her headaches were entirely gone, but her neck felt a little stiff. She returned to work on June 27, 1958, with the use of a neck collar. She gradually had a return of her previous symptoms in a lesser degree; she still had pain at the base of her neck and some pain posteriorly in the neck. There was no arm radiation of pain and no aggravation of pain with coughing or sneezing. There was no evidence of any nerve injury in either upper extremity.

On January 14, 1959, a cervical discogram performed under local anesthesia revealed that C3-4 was ruptured posteriorly, C4-5 was fused, C6-7 was normal and that the normalcy of C5-6 was doubtful. Discography was again performed at surgery on February 6, 1959, which revealed that C3-4 was definitely deranged, and C5-6 was possibly deranged. Therefore, the C3-4 and C5-6 discs were both excised and two bone plugs inserted into the area of the disc excision. The patient was maintained in a cervical neck brace for a period of approximately 2 months after her operation.

When she was last seen on April 10, 1959, her symptoms had entirely subsided. She had a solid spine fusion demonstrated by flexion and by extension x-rays, between C3-4, C4-5 and C5-6. She returned to full work activities a short time later.

Case 2 (*MR-EHF*). This 25-year-old woman was first seen in February 1957. She complained of pain in the left side of her neck, her left arm and her left upper chest. Eight months previously she had been injured while swimming. A companion standing on her shoulders had accidentally slipped and her foot slid along the trapezius muscle and laterally off the patient's shoulder. By evening she was aware of severe pain and aching in the whole left arm. Four months later she began to develop recurrent stiffness in the neck and left anterior chest pain as well as radiating pain and aching into the left middle and index fingers. The patient had had the benefit of a multitude of home, office and hospital treatments which included cervical traction, local injections, x-ray therapy, physical therapy and a variety of antirheumatic medications.

X-ray of the cervical spine revealed no gross abnormality. There was a long transverse process on C7. On examination there was tenderness of the muscles on the left side of the neck, a slight general weakness of the left shoulder girdle, and the left shoulder was carried higher than the right. Decrease in sensory perception was elicited in the left middle and index fingers. Rotation of the cervical spine was within normal limits; hyperextension caused pain in the left arm. Rotation of the head and deep inspiration obliterated the radial pulse and reproduced pain and tingling in the fingers.

Office and home treatment was continued for

FIG. 4. Cervical myelogram, major filling defect C3-4 intervertebral disc.

an additional 2 months without improvement. On March 27, 1957, the left scalenus anticus muscle was sectioned. The immediate improvement in subjective complaints after scalenotomy was short-lived. Within 4 weeks all symptoms returned. Symptomatic, sedative and supportive measures, as well as spontaneous variations, afforded only intermittent relief.

In May, 1958 the patient reported she was continuing to have distress in the left arm and shoulder girdle and now had scapular area pain. A neurologic consultant advised continued observation in the absence of objective physical findings. By the fall of 1958, the left scapular pain had become more severe and was especially distressing at night. Hyperextension of the cervical spine reproduced the pain in the left scapular area. Discograms on September 29, 1958, were abnormal in configuration at C6-7 (figs. 2 and 3). At the moment of injection of the contrast media, the patient's scapulotrapezius and left-hand pain was not only reproduced but many times intensified.

On September 30, 1958, the C6-7 disc was excised and an autogenous intervertebral body dowel fusion stabilization accomplished through the anterior approach. Postoperatively, the neck wound drain was removed in 24 hours and the sutures in 3 days. The patient was discharged from the hospital 7 days after the operation.

FIG. 5. Cervical discogram C3-4, demonstrating posterior protrusion of disc into the spinal canal

Although pain no longer disturbed her sleep, relief of subjective symptoms was incomplete. The scapular and trapezius aching and stiffness returned early in the morning. The Victoria collar aggravated her symptoms and was discarded. Three months postoperatively, the patient was subjectively improved. She had no scapular pain, no left-arm pain and no paresthesias. She required no medication for rest or activity. Six months postoperatively, biplane bending x-rays demonstrated no motion between the fused vertebrae C6-7. The patient stated she had no neck or shoulder pain and no night pain and felt she had resumed her preinjury status.

This patient's history is presented in some detail for several reasons:

1. There is no litigation or secondary monetary gain involved.

2. The mechanism of precipitation of the initial symptoms is different from the usual flexion-extension-rebound type of neck injury associated with rapid deceleration.

3. The entire gamut of symptomatic and supportive treatment measures failed over a long period of time.

4. Scalenotomy failed to give relief.

5. The symptoms in a multitude of areas, including a seeming anxiety which had continuously plagued the patient, were removed by disc removal and interbody fusion.

Case 3 (*GRC-RMS*). This 65-year-old rancher was first referred to the office on September 11, 1947, after he had been involved in an automobile accident. He received both a head and a neck injury and complained of headache on his left side, dizziness on sitting up and some blurring vision. He was considered to have a cerebral concussion and a moderately severe neck injury. He was hospitalized for 10 days and still had frontal head pain, pain in his eyes, and periods of blackout when allowed to sit up and a tendency to stagger when he tried to walk. He returned home under the care of his general physician.

He was again referred on May 9, 1958. On February 20, 1958, he had fallen off a truck while tying on a load of hay. The back of his head struck

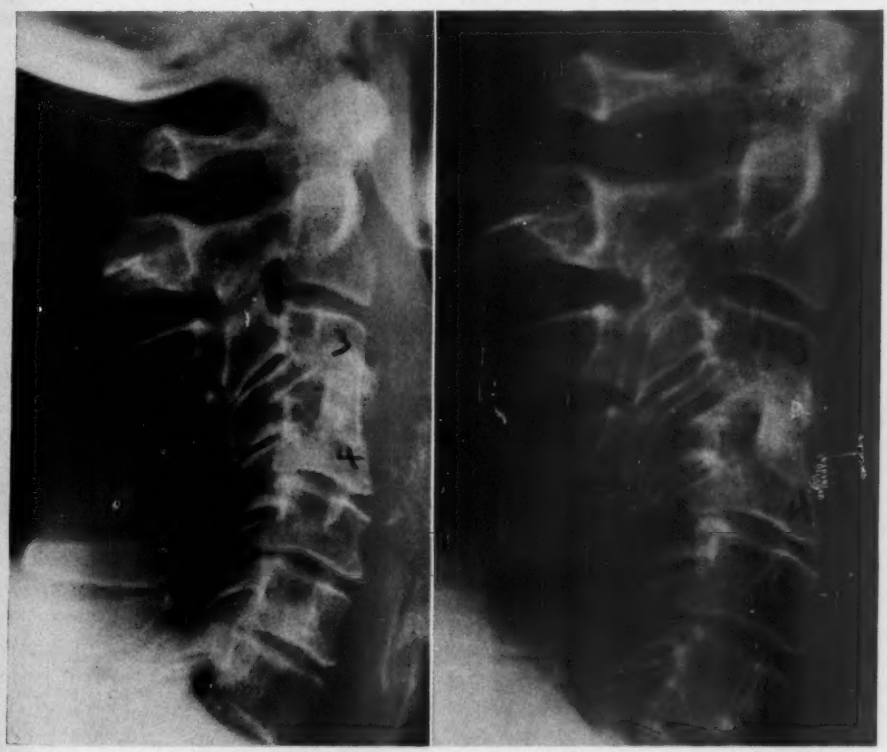

FIG. 6 (*left*). Bone graft placed between C3 and C4 vertebral bodies.
FIG. 7 (*right*). Fusion demonstrated of C3 and C4 vertebral bodies.

a bale of alfalfa on the floor, flexing his neck acutely and rendering him unconscious. He remained unconscious for about 30 min. and could not get up by himself when he regained consciousness. He said, "I couldn't move my right arm and hand at all; it seemed numb. I also had trouble moving my right foot and leg. After a while, someone came and helped me get to my feet." He was then able to get around but could not move his right side freely. Later, he was able to drive his pickup home by driving with his left foot and hand.

Function of the right leg had returned rapidly, but the right arm gained strength slowly. As he began to walk, he experienced severe pain in his right arm and leg. By the end of 2 months, pain in the right leg disappeared. The pain in the right arm was severe at times; he could get relief when he hung it over the side of the bed. The left-sided headache with pain spreading into the left eye, severe since the accident, showed very little improvement. The head pain was worse with motion, nodding or jarring.

On February 24, 1958, x-ray studies of the cervical spine showed no evidence of recent fracture. Advanced osteoarthritic changes at C4-5 and C6-7 interspaces with localized hypertrophic encroachment on the exit nerve foramina were noted. Examination on May 9, 1958, revealed slight numbness about the left eye, marked tenderness in the upper cervical region and the head slightly tilted to the left and in flexion. Neck pain was aggravated by any motion of the head. Motion was limited to 20 to 30 degrees. There was marked weakness of the right grip and slight weakness of the right leg. Sensation to pin scratch was reduced from the shoulders downward on the right side. There was a slight drift of the outstretched right arm.

On May 12, 1958, a cervical myelogram showed a filling defect at the intervertebral spaces between C2–3, C3–4 and C4–5 on the right which suggested damage or degeneration of these discs (fig. 4). On May 14, 1958, a discogram of C3–4 revealed some posterior protrusion and evidence of disc degeneration (fig. 5).

The same day at operation an incision was made in the right side of the neck. Dissection was carried along the medial border of the sternocleidomastoid muscle and the structures in the carotid sheath and along the lateral border of the trachea and esophagus to the vertebral column. The C3–4 intervertebral disc was excised. Then the nerve roots were decompressed. Two plugs of bone ⅝ in. in diameter were taken from the ilium, fashioned and driven into place in the neck.

In x-rays (fig. 6) taken on May 18, 1958, the bone graft appeared to be in good position. X-rays taken on December 1, 1958, revealed bony union had occurred between C3–4 vertebral bodies (fig. 7). The patient stated that the weakness and numbness of the right arm and hand and of the right leg and foot had disappeared. The left-side headaches had disappeared as well as the numbness about the left eye. He believed the grip in the right hand to be much improved. He had returned to his former employment as a rancher the end of the summer, 1958. Free of symptoms, the patient was discharged from further treatment.

Dr. Ralph M. Stuck
1825 Gilpin St.
Denver 18, Colorado

REFERENCES

1. Cleveland, D. A.: Management of cervical disk and cervical arthritis syndromes. Postgrad. Med., *18:* 99, 1955.
2. Cloward, R. B.: Cervical diskography. Technique, indications and use in diagnosis of ruptured cervical disks. Am. J. Roentgenol., *79:* 563, 1958.
3. Cloward, R. B.: The anterior approach for removal of ruptured cervical disks. J. Neurosurg., *15:* 602, 1958.
4. Keegan, J. J., and Garrett, F. D.: Segmental distribution of cutaneous nerves in limbs of man. Anat. Rec., *102:* 409, 1948.
5. Smith, G. W., and Robinson, R. A.: The treatment of certain cervical-spine disorders by anterior removal of the intervertebral disc and interbody fusion. J. Bone & Joint Surg., *40-A:* 607, 1958.

PLEURAL AND PERICARDIAL BIOPSY*

WILLIAM A. HOPKINS, M.D., ALFREDO ALARCON, M.D., M. BEDFORD DAVIS, M.D., AND WILLIAM C. WANSKER, M.D.

Atlanta, Georgia

Pleural effusion should no longer be considered a disease that presents a problem in diagnosis. We feel it is time to change the attitude toward this syndrome. Not too long ago all undiagnosed cases of pleural effusion were considered to be tuberculosis. We know that this is not true. Pleural effusion is not an unusual disease. It is a relatively common entity that is seen quite frequently in a general hospital. We have recently reviewed 119 consecutive patients with pleural effusion (table 1) admitted to St. Joseph's Hospital in Atlanta, Georgia, over a period of 19 months. A positive diagnosis was established in approximately 75 per cent of these patients. Twenty-nine of the 119 patients left the hospital without an adequate diagnosis being established. It is believed that by following relatively simple procedures one can arrive at an accurate diagnosis in at least 90 to 95 per cent of such patients.

In our experience the most common cause of pleural effusion seen in a general hospital is metastatic carcinoma. We were able to establish such a diagnosis without doubt in 4 patients from the pleural fluid alone, and from 23 other patients by actual biopsy. This biopsy was obtained from the pleura, scalene node or lung itself. Heart failure and pulmonary infection together made up approximately 25 per cent of the group. Pleural effusion was not an uncommon complication of heart failure. A sterile pleural effusion, which often clears rapidly and leaves no residue, may accompany pulmonary infections. It is interesting to note that during this period of observation 10 patients with pneumothorax presented a picture of associated pleural effusion. This group of patients has been discarded from our discussion. There were 5 traumatic cases, 2 spontaneous ruptures of the esophagus and 2 mesotheliomas that produced pleural effusion.

There were 5 patients with pulmonary infarcts. This group of patients often presents a difficult problem in diagnosis. Infarcts may not always be typical with sudden onset of pain, hemoptysis and a wedged-shaped shadow in the lung. The patient may begin with pain and insidious pleural effusion, and the diagnosis may remain obscure. In some patients one may have to resort to pleural biopsy.

In 119 patients there were only 7 cases established as pulmonary tuberculosis. It is possible that some of the undetermined diagnoses subsequently showed evidence of pulmonary tuberculosis. A definite diagnosis must be established in all cases of pleural effusion. We feel with relatively simple procedures the diagnosis can be established for the majority of cases. Donohoe[2] in a recent survey of the literature concluded that the diagnosis could be made in the majority of cases. Needle and surgical biopsy should be used extensively.

PLEURAL BIOPSY

Complete history and physical examination combined with adequate radiographic studies of the chest and study of the pleural fluid will establish the diagnosis in many cases. In any case of pleural effusion associated with a diagnostic problem, needle biopsy should be employed at the initial thoracentesis. This is done with the use of the Silverman needle. Multiple areas are sampled, and several biopsies of the pleural surface are obtained. We have followed the same basic technique as described by DeFrancis and associates.[1] We prefer to do the biopsy before removing the pleural effusion because this allows us a cushion between the two layers of pleura and prevents damage to the lung. At least 5 or 6 separate areas are probed with the Silverman needle, and plugs of tissue are removed for study. The fluid is then removed and thoroughly examined. This examination should consist of cultures, cell count, including differential and

* From the Thoracic Surgical Service of St. Joseph's Hospital, Atlanta, Georgia, and the Thoracic Surgical Service, Grady Memorial Hospital, Emory University School of Medicine, Atlanta, Georgia.

Presented during the Miami Beach Assembly of the Southeastern Surgical Congress March 9 to 12, 1959.

TABLE 1

Diagnosis in 119 cases of pleural effusion

Diagnosis	No. of Cases
Metastatic carcinoma	
In fluid	4
Biopsy	23
Heart failure	14
Pulmonary infection	14
Mesothelioma	2
Pulmonary infarct	5
Tuberculosis	7
Cirrhosis, liver	4
Traumatic	5
Pneumothorax	10
Ruptured esophagus	2
Undetermined	29

specific gravity. Studies for tumor cells should be done. We do not feel that the study of the sugar content of the pleural fluid is any help in the differential diagnosis of pleural effusion. If these procedures are not successful in establishing the diagnosis of the disease, bronchoscopy and scalene node biopsy should be done. If at this time an adequate explanation of the pleural effusion is not established, then open thoracotomy and pleural biopsy are definitely indicated. We feel that the above procedures other than open thoracotomy will result in approximately 75 per cent of accurate diagnosis. With open thoracotomy we feel we can establish a diagnosis in approximately 95 per cent of the cases.

The use of pleural fluid to study tumor cells is extremely difficult and hazardous. In our experience it is totally unreliable, and our pathologists feel rather insecure in making a diagnosis. Apparently the changes that go on in the mesothelial cells of the pleura in effusion can simulate malignancy; in some cases it is impossible for the pathologist to be certain. We have recently seen 3 cases, all presenting typical cells and tissue characteristic of malignancy. Yet of the 3, only 1 was proven to be malignant.

CASE REPORTS

The first patient was a 34-year-old white woman who came in with a history of pain in her chest and pleural effusion. She had a questionable mass in her left lower lobe. Bronchoscopy and scalene node were negative. The pleural effusion showed typical cells of carcinoma. Since that time the patient has died, and we have proven carcinoma. Her pleural effusion is not unlike that of the next 2 cases (fig. 1*A*).

The second patient was a 44-year-old white man who had had sudden onset of pain in his left chest. He came into the hospital with a pleural effusion. His skin test for tuberculosis was negative. The histoplasmosis text was also negative. The pleural fluid was reported as quite characteristic of malignancy (fig. 1*B*). However, we were not completely satisfied, and a pleural biopsy was done which showed absolutely no evidence of malignancy. This patient had a small peripheral pulmonary infarct.

The third patient was a 60-year-old white man who had a history of onset of gradual pain in his chest, dyspnea and fatigue. He was admitted to the hospital, and a pleural effusion was found on the left side. This pleural effusion was sampled and found to contain cells quite characteristic of malignancy (fig. 1*C*). The pathologist felt that this was unquestionably carcinoma. We were not completely satisfied with the diagnosis and went ahead with a pleural biopsy. We found a normal pleural space except for effusion. There was a small area on the periphery of the lung which on biopsy proved to be a pulmonary infarct.

Many of these cases show quite adequately that one cannot depend on pleural effusions for diagnosis. A hopeless prognosis should not be given on a benign lesion. Unless tissue is obtained by biopsy, we cannot accept a cellular diagnosis of malignancy in pleural effusion. We feel that this justifies the need for open pleural biopsy whenever we cannot accurately establish the diagnosis by needle biopsy.

We have had the opportunity of doing an open biopsy in 23 cases of undiagnosed pleural effusion (table 2). There were 5 cases of pulmonary tuberculosis diagnosed in this manner. Four cases of metastatic carcinoma were encountered. Pulmonary infarct was encountered in 2 cases. There was 1 case of malignant lymphoma. We think the most significant and important finding in this group of cases was the diagnosis of so-called "chronic pleuritis" in 9 cases. This was thought by some pathologists to be a chronic pleural inflammatory process, by others it was called a "non-specific pleuritis." Generally speaking, it presented the pathologic picture of thickening of the parietal pleura without any inflammatory reaction and without any evidence of tuberculosis. Donohue[2] feels that there is an entity of

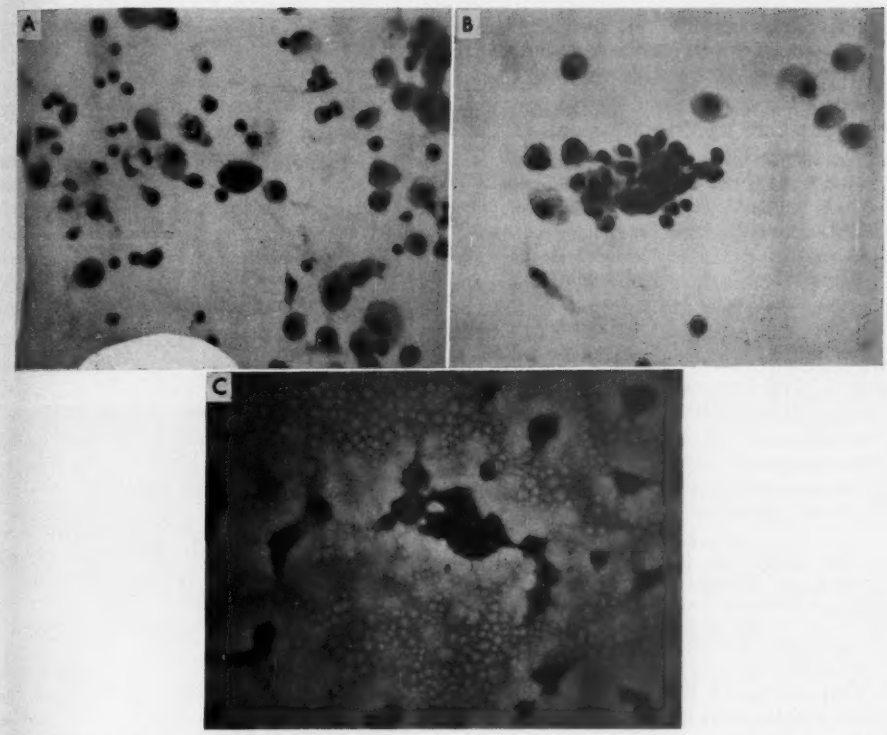

FIG. 1. Photomicrographs of cells in pleural effusion. *A*, carcinoma of lung with pleural effusion, *B*, pleural effusion with pulmonary infarct and *C*, pleural effusion with pulmonary infarct.

TABLE 2

Diagnostic results in 23 open pleural biopsies

Diagnosis	No. of Cases
Tuberculosis	5
Metastatic carcinoma	4
Pulmonary infarct	2
Lymphoma	1
Mesothelioma	2
Chronic pleuritis	9

pleuritis due to some unknown agent, probably not dissimilar to idiopathic benign pericarditis.

Multiple biopsies of the pleura were obtained in all cases. Both parietal and visceral pleura were biopsied. In some cases where there were suspicious areas in the lung, a small piece of it was removed for study. The only complication in this group was one wound infection, which cleared with adequate drainage and treatment. There were no empyemas, no deaths in the series, and no serious complications encountered.

The biopsies were obtained from the patient, in the lateral position under general anesthesia, with an endotracheal tube. A small incision was made over the 5th interspace in the anterior axillary line. A small section of the 5th rib was resected and the pleural cavity entered through the base of this rib. Adequate biopsies could be obtained from this incision. Multiple biopsies of parietal and visceral pleura were obtained. Where indicated, the lung was also biopsied. The chest was closed with thoracotomy drainage, and the tube was usually removed on the first postoperative day. The patient was up and about in 48 hr. and was usually discharged on the 5th postoperative day.

When we first began pleural biopsies we gave the patients several weeks of antituberculosis therapy, but recently we have felt that this is not necessary. If tuberculosis is encountered, we

TABLE 3

Comparison of clinical impression and actual diagnosis in 13 cases of pleural effusion

Case	Clinical Impression	Final Diagnosis
1	Tuberculosis	Chronic pleuritis
2	Carcinoma	Chronic pleuritis
3	Tuberculosis	Mesothelioma
4	Tuberculosis	Carcinoma
5	Carcinoma	Tuberculosis
6	Nonspecific inflammation	Tuberculosis
7	Tuberculosis	Carcinoma
8	Carcinoma	Carcinoma
9	Carcinoma	Tuberculosis
10	Nonspecific inflammation	Chronic pleuritis
11	Tuberculosis	Tuberculosis
12	Tuberculosis	Chronic pleuritis
13	Tuberculosis	Carcinoma

place the patient on immediate postoperative drug therapy. We have had no serious complications.

We analyzed 13 cases of pleural effusion that had no established clinical diagnosis. There was very little correlation between the clinical impression and the established diagnosis (table 3). We were unable to get an opinion in every case because often it is impossible to form even an opinion. As one can see there was only 23 per cent correlation.

We are certain that tuberculosis may be the cause of some of these chronic forms of pleuritis, and it is necessary to follow this group closely. If we cannot establish the diagnosis of tuberculosis, we do not suggest long-term chemotherapy.

PERICARDIAL BIOPSY

We feel that pericardial effusion presents the same challenge and basically the same diagnostic approach as that of pleural effusion. We have followed the same approach as the one suggested by Proudfit and Effler[3] in 1956. It appears to us to be a rational approach although we do not agree with their suggestion of a pericardial window. We do not believe that the diagnosis of idiopathic pericarditis should be entertained until all methods of diagnoses are used. It is an accepted fact that when the diagnosis of constrictive pericarditis is made, one goes ahead

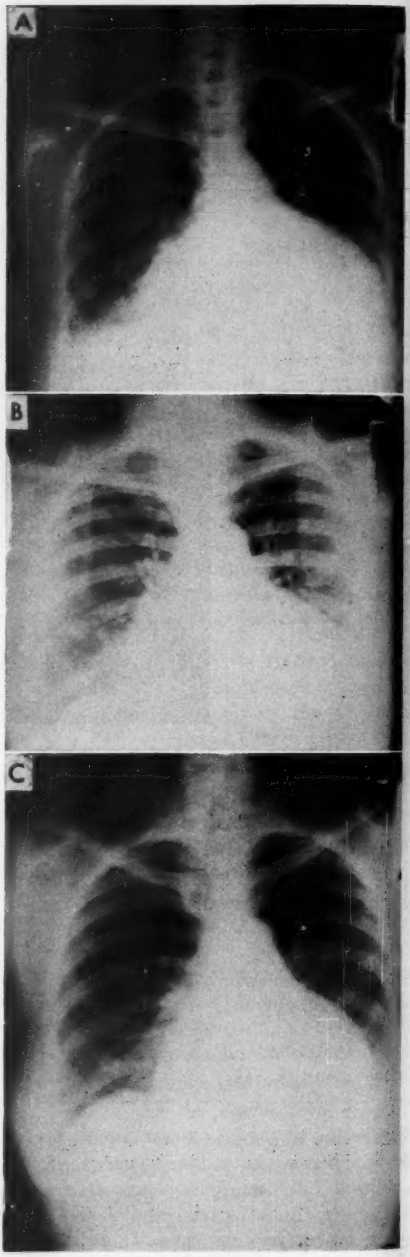

FIG. 2. Roentgenographs of chronic pericardial effusion. *A*, before biopsy and pericardial window, *B*, after biopsy, *C*, after removal of pericardium.

with surgery and decortication of the heart. This is an acceptable procedure, and should be done as soon as one can establish the diagnosis of constriction. The use of antituberculosis drugs has made this a safe procedure, even in the presence of tuberculous pericarditis. We are seeing more and more cases that can no longer be considered tuberculosis. As pleural effusion has been found not to be tuberculosis in all cases, neither is pericardial effusion always on the basis of tuberculosis. There are many cases of so-called idiopathic pericardial effusion that clear with rest and supportive measures. This group of cases should not have a biopsy.

FIG. 3. Myocarditis simulating constrictive pericarditis or chronic pericardial effusion.

There is being seen a distinct group of cases in which the pericardial effusion persists, and the patient gradually develops signs of tamponade. This may be constrictive pericarditis and not really tamponade. It has been our experience to find that this constriction quite often begins on the epicardium and visceral pericardium. In order to appreciate it as the actual constricting element, surgery is necessary. When evidence of chronic tamponade occurs, one should be strongly suspicious of constrictive epicarditis, and surgery should be instituted. In exploring pericardial effusions an adequate operation should be done the first time. Too often one will attempt to do a biopsy of the pericardium and leave a small window.[3] A small pericardial window will soon seal and be obstructed by the lung adhering to the surface. If one undertakes to do a pericardial biopsy of the patient and finds there is no element of constriction, the anterior two-thirds of the pericardium should be removed.

A 44-year-old colored woman was admitted with a history of persistent pericardial effusion over a period of several months (fig. 2). Numerous quantities of fluid had been removed from the pericardium. On several occasions air had been injected into the pericardium and revealed no evidence of tumor. A small incision was made for exploration, and the pericardium was found to be quite thickened. A small biopsy was obtained, and a small window was made in the left side of the pericardium. The patient's postoperative course was uncomplicated for about 2 months with decrease in the amount of pericardial effusion. The diagnosis was pericarditis, chronic and nonspecific. Then gradually (fig. 2*B*) the pericardial

TABLE 4

Diagnosis in 9 cases of pericardial biopsy

Age	Sex	Clinical Diagnosis	Final Diagnosis
yr.			
11	F	Chronic pericardial effusion	Myocarditis
44	M	Constrictive pericarditis	Myocarditis
53	F	Chronic pericardial effusion	Chronic pericarditis
38	F	Chronic pericardial effusion	Lymphopericardium
45	M	Tuberculous pericarditis	Chronic pericarditis
28	F	Pericarditis with effusion	Chronic pericarditis
37	M	Tuberculous pericarditis	Chronic pericarditis (probably collagen disease
28	M	Pericardial effusion	Chronic pericarditis
48	F	Constrictive pericarditis	Chronic pericarditis with effusion

effusion began to return, and she was brought back into the hospital. An adequate pericardiectomy was done. Since that time she has been well (fig. 2C).

This case illustrates the inadequacy of a small pericardial window. If one does go ahead with a pericardial biopsy, he should be prepared to do a pericardiectomy at the same time. Sampling of the fluid should be done. If the pericardial effusion is large and there is a cushion of fluid between the pericardium and the heart, a Silverman needle may be used to obtain an adequate biopsy. However, this should be approached with caution, as the myocardium itself may be harmed. In some cases the removal of fluid with replacement of air will demonstrate thickness of the pericardium as well as the actual size of the heart.

There is one condition in which pericardial exploration may be carried out without anything actually being accomplished. Occasionally a patient will be seen with a diagnosis of constrictive pericarditis. In certain cases it is extremely difficult to tell the difference between a chronic myocarditis and constrictive pericarditis. In this group of cases an adequate exploratory operation may be necessary to completely rule out the presence of chronic cardiac tamponade or constrictive pericarditis.

A 44-year-old white man had a long-standing history of edema of the extremities, chronic enlargement of the liver and increased venous pressure. Radiographic studies showed an enlarged cardiac silhouette with poor pulsations along the cardiac borders (fig. 3). After many months of indecision an exploration was done. This patient had a chronic myocarditis, and there was absolutely no evidence of cardiac tamponade or constriction by the pericardium.

We have had the opportunity to biopsy 9 cases with the diagnosis of pericardial effusion. All had signs of chronic tamponade (table 4). It is interesting to note that not a single one of these cases turned out to be tuberculous pericarditis. In only 1 case was constriction encountered at the time of surgery. This constriction was limited to the epicardium requiring a total epicardiectomy to relieve the signs of chronic constrictive epicarditis. There was one death in this group. It was an 11-year-old child with myocarditis. All but 1 of the cases had the anterior two-thirds of the pericardium removed. The one exception had a small window with subsequent pericardectomy. There were no complications in this group. It is apparent that a nonspecific pericarditis with effusion exists and must be considered in all cases where the effusion does not clear.

SUMMARY

We have reviewed 119 consecutive cases of pleural effusion admitted to a general hospital. The results of this study are tabulated. Diagnosis was established in 75 per cent of the cases. With the use of adequate history and physical examination, pleural biopsy and the study of the pleural fluid, associated with bronchoscopy and scalene node biopsy, a diagnosis can be established in approximately 75 per cent of these cases. With open thoracotomy one can extend this diagnosis to approximately 90 per cent. No case of pleural effusion should be allowed to go undiagnosed unless the condition of the patient does not warrant the necessary studies.

Pericardial effusion falls into the same category, and in the majority of patients an adequate diagnosis can be established. We feel that patients with so-called chronic tamponade due to pericardial effusion should in every instance be biopsied, and if the presence of constriction either of the epicardium or the pericardium is encountered, it should be removed. If chronic pericardial effusion is present, a pericardiectomy should be done. The use of a pericardial window should be discontinued. In certain cases of myocarditis it may be necessary to do an exploration to rule out the presence of constriction or chronic effusion.

Dr. William A. Hopkins
Thoracic Surgical Service
St. Joseph's Hospital
Atlanta, Georgia

REFERENCES

1. DeFrancis, N., Klosk, E., and Albano, E.: Needle biopsy of parietal pleura, preliminary report. New England J. Med., *252:* 948, 1955.
2. Donohoe, R. F., Katz, S., and Matthews, M. J.: Pleural biopsy as an aid in the etiologic diagnosis of pleural effusion. Ann. Int. Med., *48:* 344, 1958.
3. Proudfit, W. L., and Effler, D. B.: Diagnosis and treatment of cardiac pericarditis by pericardial biopsy. J. A. M. A., *161:* 188, 1956.

Editorial

REGRESSION IN THE TREATMENT OF MAMMARY CARCINOMA

Progress in surgery does not always move steadily forward. There are times when it suffers regression. An instance of this occurred in the treatment of appendicitis in the years before the discovery of sulfanilamide, when the death rate rose steadily to 36 per 1000. During the past decade we have witnessed a similar deterioration in the treatment of mammary carcinoma. One cause for this is that the care of patients with this disease has been taken over by unskilled operators. The stimulus for this shift of patients came from McWhirter's teaching, that a simple mastectomy plus irradiation gives results which are as good as those following a radical mastectomy. With the prestige of the Edinburgh Infirmary behind this doctrine, and the substitution of an easy operation for a difficult one, it is not surprising that hundreds of doctors began to operate on their own patients instead of referring them to competent surgeons. Following their poorly executed operations the patients were usually referred for radiation therapy to a person inadequately trained.

What is even more distressing is that many competent surgeons, either out of pessimism over the results of their radical mastectomies or from misinterpretation of McWhirter's results, changed over to doing simple mastectomies. It is to surgeons in this group that this editorial is primarily directed. Let us look into the basis for their pessimism, and then into some facts about McWhirter's publications. Poor results following radical mastectomies are due to two causes: (1) operating on patients in whom the tumor is incurable; (2) whittling down the real Halsted procedure, either from ignorance, hurry, or sheer laziness. Haagensen has statistics to show that when these obstacles are overcome one can secure a 75 per cent 5-year survival rate in patients with axillary metastases of limited extent.

The late Dr. Barney Brooks went to Scotland to ascertain the basis for the dissatisfaction with the surgical treatment of mammary carcinoma. He found that the operations which were labeled "radical mastectomies" disregarded most of Halsted's principles and were not truly radical.

There are many competent surgeons who have followed McWhirter's lead without examining his work critically. Ackerman visited the Edinburgh Clinic and pointed out the following: 13 of the 719 patients did not have carcinoma; 220 of the patients had a concomitant bilateral oophorectomy. All of the patients in whom the disease persisted after irradiation were treated by an axillary dissection or by hormones. McWhirter's claim that radiation therapy destroys the malignant cells in lymph nodes has not been demonstrated; in fact, examination of his own material disproved this claim. Ackerman concluded, "He has presented no objective evidence as to the necessity for simple mastectomy."

Even if McWhirter's thesis were correct, there are two practical problems which should deter most surgeons in the United States from following it; (1) there are insufficient well-trained radiotherapeutists; (2) there are many patients who cannot afford the time and the expense required for proper radiation therapy. It seems clear, then, that simple mastectomy plus irradiation should be reserved for the patients who are too feeble to undergo an extensive procedure and for those with incurable disease. The hard and deplorable fact is that many women with a mammary carcinoma which is curable by a Halsted mastectomy are denied their only chance of living by the substitution of a simple mastectomy and irradiation. This is indeed the reverse of progress.

WARFIELD M. FIROR, M.D.
1101 North Calvert St.
Baltimore 2, Maryland

The editors of THE AMERICAN SURGEON *will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

Textbook of Surgery, Ed. 3. Edited by H. FRED MOSELEY, M.S., D.M., M.CH. C. V. Mosby Company, St. Louis, 1959.

The new edition of this textbook is excellent not only for the practitioner of surgery but also for the student and teacher. The material coverage of this third edition is much more comprehensive than in past editions. Certain subject materials in this revision have been expanded and brought up to date. For example, significant advances have been made in cardiovascular and peripheral vascular surgery since the previous publication in 1955. To keep abreast of these rapid changes the editor has availed himself of the talents of eminently qualified individuals to add new chapters and to revise old ones.

Welcome additions to any textbook of surgery are discussions of physiology and pathophysiology of organ systems. The editor has included just sufficient coverage in these areas to give the book an academic bent. These are especially welcome to the instructor. The presence of these sections on physiology should act as a stimulant to the student mind. Improvement of this text can be attained by increasing still further the discussions of pathophysiology in disease.

This textbook is well indexed, attractively bound, printed on substantial semigloss paper, contains 1336 pages which include 738 text illustrations plus 108 color plates. Most of the plate drawings in color are by Doctor Frank H. Netter.

This edition is a well-edited, comprehensive, excellent *Textbook of Surgery*.

ARLIE R. MANSBERGER, JR., M.D.

Breast Cancer—Factors Modifying Prognosis. By A. J. DELARIO, M.D. The Macmillan Company, New York, 1959.

This is an original book in which the author's experience and the experience of noted workers in this field have been compiled in table form. There are 137 tables with all the possible information on factors which could have prognostic significance.

The first three tables refer to the prognosis in untreated breast cancer. The others refer to prognosis with different treatment approaches, extensions of the tumor, and some probable etiologic factors. He concludes with tables and comments of the results obtained in different groups by him and by other authors.

The only fault with this book, if there is any, is the analysis of his own material; some of the groups of patients are too small to have statistical significance.

This book represents a tremendous amount of work and will be of great help for the student of breast cancer problems.

FERNANDO G. BLOEDORN, M.D.

Physiology of Cardiac Surgery. By FRANK GOLLAN. Charles C Thomas, Springfield, Illinois, 1959.

The application of hypothermia to surgery has been characterized by much divergence of clinical jurisprudence and emotional appeal. The Gargantuan experimental research literature attests both to the interest and to the lack of solidarity about what hypothermia does to the homeotherm, how to use it or whether to use it at all. Dr. Gollan has provided a much needed appraisal of the matter, based largely on his own very interesting and competent studies. Written in a delightful style the author proves himself not only well versed in a highly controversial scientific area but also a master of wit. His scholarly treatment punctured with pungent philosophy has yielded an informative and entertaining document. His strong emphasis on the biologic differences between hibernation and hypothermia should serve to correct much of the confusion that exists about "artificial hibernation."

The monograph is essentially a comparative essay dealing with surface cooling versus extracorporeal cooling. Although there may exist differences of opinions regarding the physiologic changes that occur in the former instance, the author presents convincing arguments in support of blood vessel cooling, of which he has long been an enthusiastic proponent. The underlying theme of the importance of anoxia in hypothermia, regardless of the method of induction, is unchallenged. The development of necrosis as a result of hypothermia has been disputed by other reports in which no pathologic morphologic changes have been observed. The discussion concerning ventricular fibrillation and problems on rewarming are highly relevant.

The monograph, all in all, is stimulating and highly provocative. An excellent bibliography is included.

EMIL BLAIR, M.D.

Vol.

Surv
large
postop
100 pa
defects
manag
numer
assume
often
standa
assiduo
In ad
to cope

HYPE

Early
oxygena
the cen
maintain
and surg
paramet
the post
ons.
Hyper
are stat
presence
may libe
hypotens

* From
of the Al
versity o
Departme
Clinic and
Research,
Present
the Sout
Colorado,